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THE LOCAL CONTROL OF THE OCULAR CIRCULATION. FRANCIS HEED ADLER

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VOL. LIII. No. 1.

ARCHIVES OF OPHTHALMOLOGY.

THE LOCAL CONTROL OF THE OCULAR CIRCULATION.[1]

BY DR. FRANCIS HEED ADLER, PHILADELPHIA.

FROM THE DEPARTMENT OF PHYSIOLOGY, UNIVERSITY OF PENNSYLVANIA.

(With six illustrations in the text.)

MANY observers have sought to demonstrate the presence of vaso-motor nerves to the eye. As early as 1864 Adamuck (1) showed a fall in intraocular pressure when the cervical sympathetic was stimulated. Since then different observers (2), (3), (4), (5), (6), (7), (8), (9), (10), (11), (12), (13), using different technique and working on different animals, have reported widely discordant results. The literature is full of experiments improperly controlled, and far-reaching conclusions have been based on scanty experimental evidence.

In 1904 Henderson and Starling (14) gave a critical review of the literature and by a series of well controlled experiments proved that stimulation of the head-end of the cervical sympathetic or the superior cervical ganglion caused a rise in intraocular pressure on the side stimulated. This rise was of sudden onset and short duration, and was followed by a gradual and more or less persistent fall below normal. These changes in intraocular pressure were independent of any changes in blood

[1] Read before the Section on Ophthalmology of the College of Physicians, March 15, 1923.

pressure, which remained constant throughout the experiment. They proved that the initial rise was due to the contraction of unstriated muscle fibers surrounding the globe in Tenon's capsule, which fibers are supplied by the sympathetic, since the rise occurred synchronously with retraction of the nictitating membrane and protrusion of the globe from the socket, and occurred after the animal had been bled to death. The fall in pressure they attributed to a constriction of the intraocular blood vessels, through the agency of vaso-constrictor fibers in the sympathetic. All observers who have since noted this fall in pressure attribute it to vaso-constriction, and likewise draw the conclusion that the eye along with the rest of the head area is supplied with vaso-constrictor fibers through the sympathetic system.

No attempt has been made, as far as we are aware, to prove this conclusively, although several observations lend it support. In 1912, however, Hill and Flack (15) made the following objections to Henderson and Starling's conclusions: "Starling and E. E. Henderson ascribe the rise to contraction of the orbital muscle and the fall to vaso-constriction of the intraocular arteries. Dr. W. T. Lister examined the eye of one of our cats with the ophthalmoscope and was unable to note any constriction of the retinal vessels during stimulation which produced full dilatation of the pupil. We would point out that dilatation of the pupil must cause expulsion of blood from the iris and that the volume of blood so expelled must be made good either by a secretion of aqueous or by expansion of other blood vessels, *e.g.*, in the ciliary region. The latter probably takes place, and as the dilating iris shrinks the veins which drain it dilate in the ciliary region. We have no evidence to offer as to how the small changes of intraocular pressure occur during the stimulation of the cervical sympathetic."

A further objection to concluding the presence of vaso-constrictor fibers from a fall in intraocular pressure lies in the fact that the previous rise in pressure caused by a compression of the globe by the unstriated muscle might well squeeze fluid out through the filtration spaces. When the compression ceased there would be a diminished volume of fluid in the eye, which would reveal itself in lowered intraocular pressure, and

which would remain until the amount of fluid lost was made up from the blood stream. We see this clinically as the result of massaging the eyeball. An abnormally high tension can be reduced several millimeters of mercury by massage and Magitot and Bailliart (16), (17) have shown that simple pressure applied to the globe can reduce the intraocular pressure to zero. In the use of the tonometer we see this effect, as repeated applications of the instrument frequently give lower readings.

It seemed advisable, therefore, to attempt a series of experiments which would show conclusively the direct effects on the ocular blood vessels of stimulating the sympathetic, and with this in view we applied a method we had been using for the study of the formation of fluid in the eye, which was suggested to us by Dr. Merkel H. Jacobs. By means of an apparatus which we will describe we have found that by raising the general blood pressure and keeping the intraocular pressure constant at its previous normal level we obtain a rapid formation of fluid in the eye. This fluid is not normal aqueous, but contains a high percentage of albumen and occasionally shows red blood cells under the microscope. It is due to a hyperæmia of the blood vessels, caused by increasing the normal difference which obtains between intravascular and extravascular pressure. If we maintain this new difference of pressure at a constant level we find that the rate at which the fluid is formed in the eye becomes constant. Under such conditions the rate at which fluid is formed will vary with the caliber of the blood vessels. By carefully controlling all other factors, then, if we can show a diminished rate of fluid formation when the cervical sympathetic is stimulated, we are justified in attributing it to a vaso-constriction of the intraocular blood vessels.

DESCRIPTION OF APPARATUS.

The intraocular pressure is measured by a method somewhat similar to that used by Henderson and Starling (14), keeping a small air bubble at a meniscus so that no fluid either enters or leaves the eye. In the diagram (see Fig. 1), A is a hollow sharp needle which is pushed through the cornea into the

anterior chamber in such a manner that no fluid escapes and the iris and lens are not touched. The needle is attached to a stop-cock, B, by means of heavy rubber tubing, which in turn is attached to a piece of straight glass tubing. The whole system is filled with normal salt solution and the cock closed as shown in the diagram. This is then detached from the straight glass tube, C, and the needle inserted in the anterior chamber. The glass tube C has three outlets two of which are

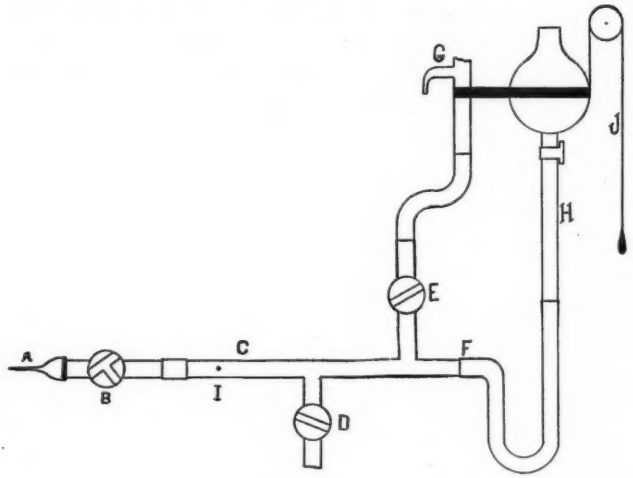


FIG. 1.

guarded by stop-cocks. The outlets are designated D, E, and F. The outlet D is for the introduction of an air bubble. E connects by rubber tubing with a vertical glass tube attached to the pressure-bottle, H. A small horizontal side tube comes off this tube and is bent down at a right angle and open to allow fluid to drop from it. The opening, F, is connected by rubber tubing to the pressure-bottle, H, which can be raised or lowered to alter the pressure in the system, and is counterweighted over a pulley to allow free motion. When the needle has been placed in the eye the connection is made between B and C, and the air which has been introduced by this manipulation is washed out of the side tube of B. A small air

bubble is then introduced at D and carried down the glass tube to a mark at I. By closing off the pressure-bottle and opening E the zero reading of the instrument can now be obtained. The cocks G and D are then closed. H is opened and the pressure-bottle raised until it approximates what the intraocular pressure should be, as determined by past experience of the usual relation between it and the general blood pressure. The cock at B is then turned so as to connect the eye with the system. If the pressure-bottle has been set at just the right height the air bubble will remain at its meniscus I, pulsating back and forth with each beat of the heart. If the pressure in the eye is higher than that in the system the bubble will start moving up the glass tube towards the pressure-bottle, which must immediately be raised until the bubble remains fixed at its meniscus. It the bubble starts moving in the opposite direction then the pressure in the system is higher than that in the eye, and the pressure-bottle must be lowered accordingly. In this way the eye and the system are brought into equilibrium of pressure. If E be now opened the height of the column of salt solution above the zero mark gives the pressure in the eye in millimeters of water (normal salt solution). The tube G is now raised or lowered until the top of the column of water in it comes level with the horizontal side tube. This side tube is quite short, 1.5*mm*, and as described is bent down at a right angle and open to allow fluid to drop from it. Every drop of fluid coming from the side tube falls on an electrical drop recorder (18), which records by a signal magnet on the smoked drum. The pressure-bottle being closed off the intraocular pressure is balanced by the column of water in the tube G. By this method the pressure in the eye is kept from increasing above its determined value.

As long as the blood pressure remains constant the intraocular pressure remains constant. The blood pressure is measured in the opposite carotid or subclavian artery, using a mercury manometer with a float which records on the smoked drum. By tightening up on a loose ligature placed around the abdominal aorta just above where the superior mesenteric artery comes off the blood pressure is suddenly raised. Since the intraocular pressure is kept at its previous level, whereas the blood pressure in the intraocular blood ves-

sels is raised, we obtain an increased difference in the pressure normally existing between the two sides of the vessel walls, and due to the hyperæmia so created blood serum is poured out from the vessels. This displaces fluid from the apparatus and is manifested by drops coming from the horizontal side tube above described. After some oscillations the blood pressure steadies down to its new level and the rate of formation of fluid in the eye becomes constant, as evidenced by a constant rate of dropping from the tube. It must be understood that no claim is made that the fluid being formed is normal aqueous and that we are estimating the rate of formation of aqueous humor. This fluid was shown to be comparable to blood serum in its albumen content. When the rate of formation of this fluid has become constant the head end of the cut cervical sympathetic on the side of the eye experimented is now stimulated by a faradic current, and the effects noted on the rate of formation of fluid.

For the proper understanding of the results obtained by this method it is necessary to emphasize the following:

1. The intraocular pressure is never allowed to change from its original level. The new-formed fluid, which in the intact eye would raise the intraocular pressure until it inhibited further fluid formation, is allowed to escape. Hence the amount of fluid formed at a given elevation of blood pressure is no measure of the amount of fluid formed at that pressure in the intact eye.

2. The method does not measure the total rate of formation of intraocular fluid, since some may be escaping by way of the filtration angle. It has been shown, however, that if the pressure in the anterior chamber be kept constant the rate of absorption of fluid from the anterior chamber is constant. [Henderson and Starling (19), E. Seidel (20)]. Since the intraocular pressure is kept constant throughout the experiment it follows that the escape of fluid from the anterior chamber remains constant, and that any changes in rate of flow observed under stimulation of the sympathetic are not due to a changed rate of absorption of fluid from the eye.

3. Since stimulation of the cervical sympathetic at first causes a rise in intraocular pressure due to compression of the globe by the unstriated muscle we found it necessary to run

two sets of experiments—a. In which Tenon's capsule is left intact. b. In which an attempt is made to free the globe from any compression by cutting completely through Tenon's capsule.

EXPERIMENTAL DATA.

Cats were used in all experiments, and were anæsthetized with ether, and then given urethane by stomach tube, after which the ether was withdrawn. Protocols representative of all the experiments done are given below.

11-11-22

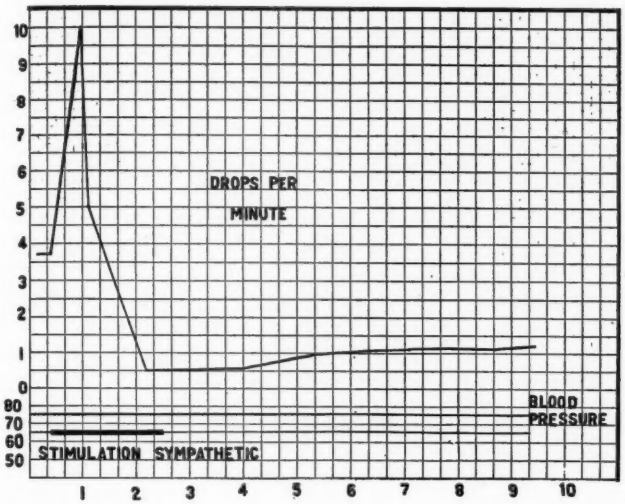
Cat, anæsthetized with ether, and given urethane by stomach tube. Ether withdrawn. Blood pressure measured in right carotid artery. Intraocular pressure measured in left eye. Both vagi cut to avoid depressor reflex when blood pressure is raised. Left cervical sympathetic cut and head end placed on platinum electrodes. Loose ligature thrown around abdominal aorta above the origin of the superior mesenteric artery. By careful dissection Tenon's capsule was cut through all around the globe, thus freeing this eye from any compressing influence of its unstriated muscle. After the intraocular needle had been satisfactorily inserted and time allowed for the eye to quiet down the intraocular pressure was measured as described above, and the vertical tube set at a height so that the top of the column of salt solution came level with the horizontal side tube opening. The blood pressure measured 50*mm* Hg (mean pressure) and the intraocular pressure 15*mm* Hg (200*mm* water). The ligature around the aorta was now tightened and the blood pressure immediately rose and reached a constant level of 116*mm* Hg after a slight secondary fall. Coincidently with the rise of blood pressure drops started falling from the side tube. When a fairly constant rate had been established the cervical sympathetic was stimulated. The drops immediately began falling at longer intervals, and only gradually returned to near their previous rate. During the stimulation there was marked dilatation of the pupil,

which lasted only as long as the stimulus was applied. From the table it can be seen that with the blood pressure remaining constant the rate of formation of fluid fell from one drop every sixteen seconds to one drop every forty-two seconds, or nearly one third as fast. It will be noticed that this rate does not come back to normal, and that this is probably due to a gradual fall in blood pressure which came about after the maximum decrease in rate of fluid formation had made itself manifest. During the stimulation there was no increase in the formation of fluid, nor was there any increased rate of dropping from the side tube as might be caused by the compression of the unstriated muscle in Tenon's capsule. As will be seen in the next protocol if Tenon's capsule is left intact this preliminary increased rate of dropping occurs only during the application of the stimulus, if this be short, or only through the first part of it if it be long continued. In other words, it represents the amount of fluid being squeezed out of the eye by compression of the unstriated muscle.

TABLE NO. 1

Intraocular pressure maintained at 15*mm* Hg

Time	*Interval between drops*	*Blood pressure*	*Procedure*
0″	0″	50*mm* Hg	
14″	14″	115	Aorta ligated
28″	14″	116	
43″	15″	"	
58″	15″	"	
1′14″	16″	"	
1′31″	17″	"	
1′48″	17″	"	
2′4″	16″	"	Stimulate cervical
2′20″	16″	"	sympathetic 1′27″
2′47″	27″	118	
3′19″	32″	"	
4′1″	42″	"	
4′34″	33″	"	
5′10″	36″	"	
5′45″	35″	"	
6′24″	39″	"	
6′59″	35″	"	
8′22″	25″	112	



RECORD No. 1. 11–11–22.

Intraocular pressure left eye. Tenon's capsule cut through completely. Blood pressure measured in right carotid. Stimulation of left cervical sympathetic.

11–21–22. See Table No. 2, Record No. 3.

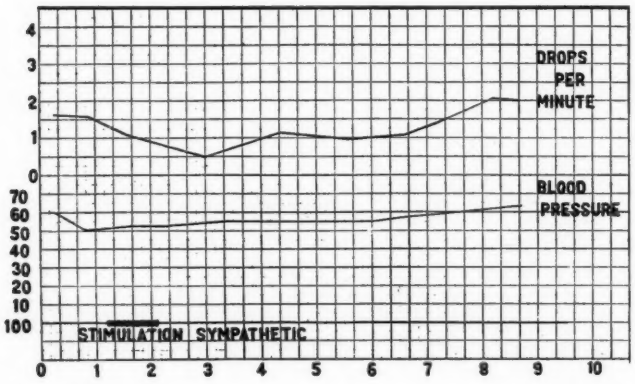
Cat prepared in the same manner as preceding with the exception that Tenon's capsule was left intact. The blood pressure at the start was 118*mm* Hg. The intraocular pressure measured 27*mm* Hg. On clamping the aorta the blood pressure rose to 180*mm* Hg and after oscillating reached a constant level of 150*mm* Hg. As soon as the blood pressure was raised drops started coming from the side tube, and gradually assumed a constant rate of one drop every 29 to 33 seconds. The cervical sympathetic was then stimulated with faradic current. Two drops came from the tube at close intervals, and then the rate fell very markedly. During the stimulation the pupil dilated widely, but immediately returned to its previous size when the stimulus was withdrawn. The rate of formation of fluid then gradually returned to normal. The two drops coming in rapid succession are due to the compressing action of the muscle in Tenon's capsule, as is proved by the

time they occur, and by the fact that they do not last through the whole time the stimulus is applied; in other words they simply represent the amount of fluid squeezed out of the eye by the muscle contracting. In part of the record not shown we found that gentle pressure on the globe with an instrument produced identically the same results. The blood pressure remained constant throughout the experiment.

TABLE NO. 2

The intraocular pressure was maintained at *27mm* Hg throughout the experiment.

Time	*Interval between drops*	*Blood pressure*	*Procedure*
0	0	118*mm* Hg	
29″	29″	150	Cervical sympathetic stimulated
58″	12″	"	
1′10″	9″	"	
1′19″	56″	"	
2′15″	28″	"	
2′43″	22″	"	
3′5″	24″	"	
3′29″	25″	"	



RECORD NO. 2. 11–11–22.

Intraocular pressure left eye. Blood pressure right carotid artery. Both vagi cut. Loose ligature around aorta. Tenon's capsule cut through completely. Stimulation left cervical sympathetic.

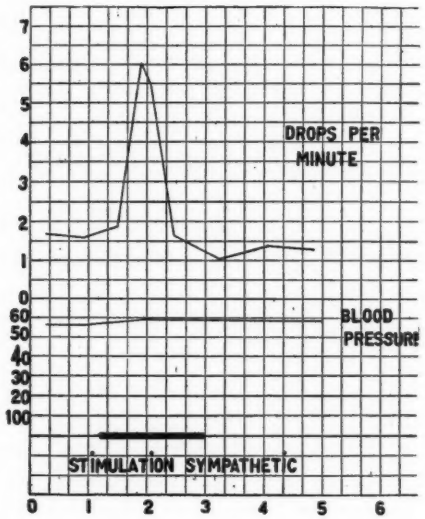
In the first record with Tenon's capsule cut the effect of stimulating the cervical sympathetic was to decrease the rate of formation of fluid. Since the blood pressure and the intraocular pressure have remained constant through the experiment the most plausible explanation of the delayed rate we believe to be a narrowing of the intraocular blood vessels—whether arterioles or capillaries or both we cannot conjecture. The second record shows the effect when Tenon's capsule is intact and supports Henderson's and Starling's view that the initial rise in pressure on stimulating the sympathetic is a compression of the globe by the unstriated muscle fibers.

In several articles explaining the cause of the increased albumen content of the aqueous after puncture of the anterior chamber Wessely (13), (21), (22) showed that by the subconjunctival injection of adrenalin or by prolonged faradization of the sympathetic he could reduce the amount of albumen, from which he concluded that the high albumen content after puncture was due to a hyperæmia of the intraocular vessels. These experiments lend further support to Wessely's conclusions.

Asher (23) in 1912 found that extirpation of the superior cervical ganglion delayed the appearance of fluorescine in the anterior chamber and lowered the albumen content of the aqueous. He attributes this to a decreased permeability of the intraocular blood vessels after removing sympathetic impulses to the eye. He maintained, therefore, that the sympathetic affords a tonic influence on the permeability of the vessels, in the sense that the permeability is increased by its presence. This would mean that by stimulating the sympathetic the permeability should be still further increased—a fact to which we cannot subscribe in the light of observations by numerous investigators besides ourselves. Asher does not clearly state whether sufficient time had elapsed after extirpation of the ganglion for degeneration of the nerve fibers to take place, and it is possible that in his section he is simply obtaining a mechanical stimulation of the nerve. If this is so his conclusions are unfounded and would show instead that when the sympathetic is not functioning the permeability is increased, which accords with our views, and would be suggestive evidence that normally the intraocular vessels are kept in

a somewhat tonically contracted state by the action of the sympathetic.

In 1918 R. H. Kahn (24) published a paper to support Hamburger's views on the existence of a water-tight junction



RECORD No. 3. 11–21–22

Intraocular pressure left eye. Blood pressure right carotid. Both vagi cut. Loose ligature around aorta. Tenon's capsule intact. Stimulation left cervical sympathetic.

between the posterior surface of the iris and the anterior surface of the lens. We were astonished to find a record in Kahn's paper which seemed to be an exact duplicate of the records we have obtained in our present experiments. Kahn's method was entirely different from ours, and his point of attack was from a different angle. It would be out of place in this article to go into the details of Kahn's work and an analysis of his results. They have already been ably criticized by Seidel (25) and both should be read in the original for a full account. It is impossible for us to apply Kahn's conclusions to our own

experimental data, since he gives no blood pressure records with his evidence, and since he did not actually measure intraocular pressure, but merely set his instrument at a height which he judged the intraocular pressure to be. His conclusions were that the dilatation of the pupil was the essential and primary factor in the results he obtained. Since Kahn's experiments were so poorly controlled and were based on the assumption that the sympathetic contained vaso-dilator fibers—an assumption for which there is absolutely no experimental proof—it was impossible to apply them in a critical way to our own. We found it necessary, therefore, to repeat our own experiments with the addition of having the pupil widely dilated before we stimulated the sympathetic.

For this purpose we used a 2% solution of atropine sulphate, and secured maximal dilatation of the pupil by 4–6 drops five minutes apart. The experiments were carried out in every detail as those previously described.

EXPERIMENTAL DATA.

1. We obtained the same results as before with a pupil widely dilated before stimulating the cervical sympathetic. When the nerve is being stimulated with a strong current there is a very slight further dilatation than that produced by the atropine, as stated by Cushny (26) but is negligible compared to that produced by the atropine and could not influence our results.

2. The effect of compressing Tenon's capsule is intensified under atropine, *i.e.*, more drops come off during the early part of the stimulation when the unstriated muscle has not been cut. This is probably due to the iris being folded up in the filtration angle of the eye, blocking to some extent the outflow of aqueous when the globe is compressed. It showed us, further, that many times when we thought we had completely destroyed all effects of the muscle on the eye we were mistaken. When, however, no increased rate of dropping occurred at the beginning of stimulation we regarded this as sufficient evidence that for our purposes at least the compressing effect was negligible, if not proved entirely absent.

3. The conclusions drawn by Kahn from his records are

erroneous. We cannot offer any suggestions as to the proper interpretations of his experiments in the absence of sufficient experimental data and proper controls.

4–12–23

Cat. Anæsthetized and prepared as in preceding experiments. Tenon's capsule not cut through. 6 drops of a 2% solution of atropine instilled in left eye. Pupil widely dilated. Blood pressure right carotid artery. Intraocular pressure left eye. Stimulation of left cervical sympathetic.

See Record No. 5.

Intraocular pressure maintained at 30*mm* Hg.

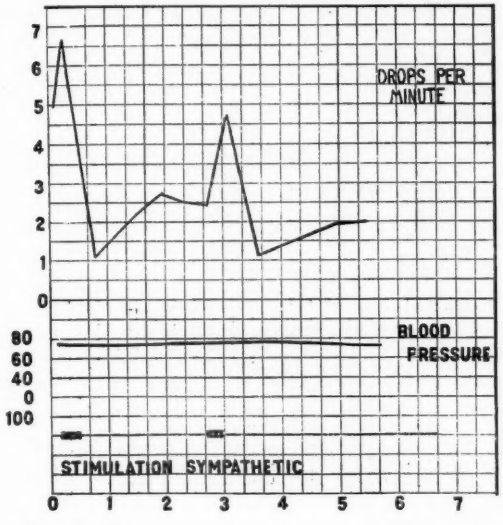
TABLE No. 3

Intraocular pressure maintained at 30*mm* Hg.

Time	*Interval between drops*	*Blood pressure*	*Procedure*
0	0	125*mm* Hg	
1′	14″	160	Aorta ligated
1′15″	15″	"	
1′30″	15″	"	
1′47″	17″	"	Stimulate cervical
1′51″	5″	"	sympathetic 1′41″
1′56″	5″	"	
2′7″	11″	"	
4′7″	120″	"	
5′38″	91″	"	
6′42″	64″	"	
7′33″	51″	"	
8′23″	50″	"	
9′11″	48″	"	
9′57″	46″	"	
10′39″	42″	"	

Here again we see the primary effect of compression of the globe by the unstriated muscle, followed by a long delay in the rate of formation of fluid, showing that full dilatation of the pupil does not influence the effect previously noted, and in no way can account for it.

At the end of this experiment the animal was bled to death. As soon as the blood pressure had fallen to zero a new level of intraocular pressure was read, and the instrument adjusted



RECORD No. 4. 11–21–22.

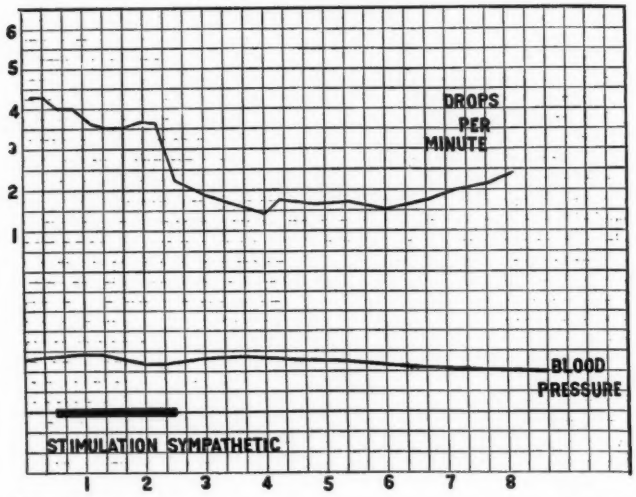
Intraocular pressure left eye. Blood pressure right carotid. Tenon's capsule intact. Loose ligature around aorta. Both vagi cut. Stimulation left cervical sympathetic. Two short stimuli given.

accordingly. The sympathetic was then stimulated. Record No. 5 shows the effect of squeezing fluid out of the eye, and compares with the records of Henderson and Starling taken similarly but measuring pressure changes instead.

SUMMARY.

A method is described by which the rate of formation of new-formed fluid in the eye can be measured. The formation of this fluid depends on increasing the difference in pressure normally existing between the blood in the intraocular blood vessels and the aqueous humor. When the rate has become constant we have shown the effects on it of stimulating the head end of the cut cervical sympathetic.

When Tenon's capsule is intact we usually see a momentary increase in the number of drops coming from the side tube. This is not seen when the muscle fibers in Tenon's capsule have



RECORD NO. 5. 4-12-23.

Left pupil widely dilated with atropine sulfate prior to experiment. Intraocular pressure left eye. Blood pressure right carotid. Both vagi cut. Loose ligature around aorta. Stimulation left cervical sympathetic. Tenon's capsule intact.

been severed. We must conclude this to be additional evidence to that already produced by Henderson and Starling that the rise in pressure obtained by them is due to compression of the globe by unstriated muscle fibers in Tenon's capsule. In every case, whether Tenon's capsule is intact or has been cut through, stimulation of the sympathetic produces a marked decrease in the rate of formation of fluid. Since the pupillary dilatation passes off some time before the decreased rate of formation of fluid begins to be evident, and since we have obtained the same results with the pupil widely dilated from atropine before stimulation is begun, we must assume Hill and Flack's suggestions invalid.

Since the intraocular pressure remains constant throughout the experiment the rate of absorption of fluid from the eye must be constant, and so the observed effects cannot be attributed to a change in the rate of absorption of fluid due to the stimulation of the sympathetic.

Since the blood pressure during the experiment has remained practically constant we conclude that the decreased rate of formation of fluid is evidence of a constriction of the intraocular blood vessels.

In concluding I wish to express my thanks to Dr. H. C. Bazett for many helpful suggestions and constant encouragement, and to Mr. William McClenahan for his valuable assistance.

CONCLUSIONS.

1. A rise of blood pressure causes an increased formation of fluid in the eye when the intraocular pressure is maintained at its previous normal level. This is due to a hyperæmia of the intraocular blood vessels, allowing blood serum to come through into the aqueous.

2. The rate of formation of this fluid is diminished by stimulation of the cervical sympathetic;—a fact which can only be adequately explained by constriction of the intraocular blood vessels,—thus affording more direct evidence than that already produced of vaso-constrictor fibers to the eyeball by way of the cervical sympathetic.

3. There is no evidence of vaso-dilator fibers to the eyeball.

4. The effects on the unstriated muscle in Tenon's capsule, as explained by previous workers, are confirmed.

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BLINDNESS OF OBSCURE NASAL ORIGIN.[1]

By Dr. WILLIAM HUMES ROBERTS, Pasadena, California.

(With one illustration in the text.)

IT has long been recognized that varying degrees of loss of vision, up to complete blindness of one or both eyes, can be caused by readily diagnosed disease of the accessory sinuses producing a retrobulbar neuritis. To call attention to the fact that the same eye condition is frequently the result of obscure nasal disease, is the object of this paper.

A number of authors, notably Van der Hoeve (1), Sluder (2), L. E. White (3), (4), (5), (6), Cutler (7), Warlow (7), Vail (8), Thomson (9), Dunn (10), Bordley (11), Ellet (12), Jonathan Wright (2), Alan C. Woods (10), Gradle, Posey, Stark and many others have already called attention to this.

These patients are, as a rule, first seen by the oculist on account of rapid loss of vision, usually in one eye. On referring them to the rhinologist for his examination, the report usually comes back "nasal findings negative." This then is the type of case I wish to consider in this paper, for upon our careful study and prompt diagnosis may depend our patient's vision in the affected eye or eyes. A few cases tend to spontaneous recovery, but waiting for this to occur is a dangerous procedure, and one not to be recommended.

ETIOLOGY.

When we study the anatomy of the optic nerve, particularly in its relation to the posterior accessory sinuses, we are at once

[1] Read before the Pacific Coast Oto-Ophthalmological Society, June 21, 1923.

impressed with the great variations that occur. Onodi in his Atlas published in 1911 says "our observations have shown that the wall between the last ethmoid cell and the canalis opticus is nearly always as thin as tissue paper; dehiscences in the walls of the accessory cavities have been found, and there the diseased mucosa may come into direct contact either with the dura mater or the optic nerve sheath." He has shown that the optic nerve occasionally lies exposed in the sphenoid sinus for a centimeter or more.

The investigations of Odoni, Loeb and Schaeffer have shown that the vulnerability of the optic nerve, as considered in the present paper, lies in its close relationship to the sphenoidal sinus and the posterior ethmoid cell.

Vail (8) and Sluder (2) refer to hyperplastic changes found in the ethmoid and sphenoid. To quote Vail: "hyperplasia is defined as an increase of the fibrous connective elements of living anatomic structures due to a low grade of localized inflammation. . . . Hyperplasia is a nonsuppurative process. . . . Hyperplasia of the ethmoid bone is a rarefying osteitis associated with inflammatory swelling and fibrous thickening of the mucous membrane lining its cavities, causing œdematous tissue to fill them without the formation of pus." Hyperplasia in itself amounts to little but when to it is added an infection with extension to the optic nerve, its trivial nature at once disappears.

Jonathan Wright in his introduction to Sluder's work on *Headaches and Eye Disorders of Nasal Origin* says (pages 23 and 24), "It would probably be difficult to find an adult individual in temperate or cold climates who does not present an example of this bone change within his nasal chambers, which we have a right to call pathological. It is only exceptionally that the symptoms to which it gives rise are sufficient to cause him to seek relief. When, however, it causes bony occlusion of a nasal fossa by means of a spur or deviation, when it stops up the ostium of an accessory nasal sinus and prevents proper drainage, when it impinges on a branch of a sensitive nerve or involves its peripheral distribution, marked symptoms and a long train of consequences may ensue. . . . When in the walls of the sphenoidal or ethmoidal sinus there is involvement of the optic nerve, in so far as it depends on

bony pressure, blindness, partial or complete, is pretty sure to occur. When, however, the alarming symptoms of optic involvement are recent and slight, the trouble may not be due to a bony pressure, but to a pressure of soft parts, or to an extension of their inflammation or of their vascular congestion. These latter conditions may be relieved by giving free drainage and ventilation to an occluded sinus; but in an inaccessible region, if there is pressure of a bony surface, such as the swelling, upon a sensitive or an optic nerve, it is difficult to see how the symptoms are to be relieved. Fortunately, there is good reason to believe that in the nature of things, the encroachment of the field of engorgement and soft hyperplasia upon the nerve structures gives a timely warning so that surgical interference is possible before an irreparable condition results.

"It is perhaps well to remark that we have sought some objective evidence of inflammation spreading along the sheath of the optic nerves from the foci of inflammation in the sinuses. Though there is clinical reason to suppose this sometimes occurs, we have not as yet secured objective proof of it in the material at our disposal."

The impairment of central vision is due to a retrobulbar neuritis, the papillomacular bundle being particularly vulnerable, the result usually of inflammation of that part of the optic nerve lying in the bony optic canal.

Since visual disturbance—impairment of central vision, blindness, and organic optic nerve changes—are produced by accessory sinus disease, we *must* remember that this is but one of many causes. I earnestly caution you against making a hasty diagnosis. Bear in mind that brain tumors, organic brain disease, pituitary disease, multiple sclerosis, toxic amblyopias, toxæmias, diabetes, syphilis, and the optic nerve changes due to primary eye diseases, all cause loss of vision. These must *all* be ruled out before attempting intra-nasal surgery. An operation on the sinuses for the relief of blindness caused by any of the aforementioned diseases, will not only *not* relieve the progressing loss of vision, but bring into disrepute intra-nasal operations and possibly prevent their employment in cases in which they are urgently required.

DIAGNOSIS.

When as great an authority as Van der Hoeve (1) says, "The ophthalmologist has in the eye no sign at all to distinguish the origin of a retrobulbar neuritis. The rhinologist cannot say with absolute certainty that a person has no sinus affection," we can appreciate the difficulties confronting us and the necessity of making a careful study of each case.

The ophthalmoscope shows no changes at all or at most ones that are insignificant; after subsidence of the disease atrophic changes frequently are seen.

A painstaking examination of the visual field is of utmost importance as usually there can be found a relative, and frequently a central scotoma. Measure carefully the size of the blind spot. For this it is necessary to use some form of tangent plane, Peter's campimeter, or Lloyd's stereo-campimeter. If there is enlargement of the blind spot (Van der Hoeve's symptom), but no involvement of the point of fixation, we probably have to do with a posterior sinus disease, if by the ophthalmoscope we can eliminate organic changes at and around the nerve (such as high myopia, glaucoma, opaque nerve fibers and choked disk). If the scotoma includes the point of fixation as well as the blind spot, we must then differentiate sinus disease from disseminated sclerosis and toxic amblyopias both of which usually begin with a central scotoma. The scotomas of pituitary disease vary greatly and are not always of the types seen in text books. This subject is well covered in an excellent article by De Schweinitz and Halloway (13). In central scotoma due to disease of the optic nerve, there is no change in the shape or size of objects in and about the scotoma (retinal metamorphopsia) and red and green are the first colors to be lost. In primary macular disease there is usually retinal metamorphopsia and blue is the first color to disappear. We should use the tonometer, test the corneal reflex and in other ways exclude glaucoma, as three of its symptoms, pain, sluggish pupils and paracentral scotoma, are also seen in the disease under consideration.

The ophthalmoscope should also enable us to exclude choked disk of brain tumors, as there is neither elevation of the disk, nor inflammation of the nerve. Cutler (7) has written a most

excellent paper on "The Optic Nerve in Sinus Disease," which you are advised to read. The pupil in the affected eye may be slightly enlarged and a trifle sluggish in its reaction to light. There may be slight exophthalmus. In frank cases of accessory sinus disease, transillumination and the X-ray are confirmatory; but in the obscure cases under consideration the latter only is of use and its findings must be very carefully studied and interpreted.

SYMPTOMS.

The chief complaint is rapidly developing loss of vision in one or more rarely, both eyes. There may be some discomfort in the eye, or the external ocular muscles may feel lame on moving the eyeball. There may or may not be a parietal or occipital headache. Nasal symptoms may be entirely lacking or only those of a "head cold."

EXAMINATION OF NOSE AND SINUSES.

These cases may show no nasal pathology. As a perfectly normal nose is hard to find, the slight departures from normal that may be seen in such patients do not make much impression on the examiner. A high posterior deflection of the nasal septum with some hypertrophy of the middle or superior turbinate may be noticed. The size and position of these bodies is of great importance, for if there is any interference with the ventilation or aeration of the posterior sinuses there is sufficient pathology to cause a retrobulbar neuritis on slight provocation. It is because no two rhinologists see alike on this particular point in an intra-nasal examination that we so frequently get from them the report of "negative nasal findings."

PROGNOSIS.

The prognosis is favorable if seen early. If late, it depends upon the damage done to the nerve and to the virulence of the infection.

TREATMENT.

This is surgical and consists in uncapping the posterior ethmoid cells and in opening the sphenoid sinus on the side or sides involved. Radical work on these sinuses is not necessary.

I also wish to make a plea for exploratory operation in those cases in which, by exclusion, we are forced to the conclusion that sinus disease, even though none can be demonstrated, is the cause of the trouble. In the class of cases covered in this paper, no pus nor evidence of disease may be found at operation. In the case I wish to report, ventilation, rather than drainage seemed the essential thing secured.

REPORT OF A CASE.

Mrs. C. D. in August, 1917, had a submucous resection of her septum to relieve difficulty in nasal respiration. The right middle turbinate was removed also as it was tightly adherent to the septal wall, and was diseased. From this operation she made a good recovery and has had no further nasal symptoms.

On October 8, 1922, over five years after the above operation she consulted me giving the following history:

Five days previously the right eye became a little sore. It seemed as though the external eye muscles, rather than the eye itself, were involved. Three days later, or two days before I saw her, she noticed that she could not see well with her right eye either for distance or for near.

O.D.V. $\frac{1}{10}$. O.S.V. $\frac{6}{6}$–4.

Pupils react, right may be a trifle sluggish. Media clear. Tension normal. Disks normal. Vessels normal. Fields:

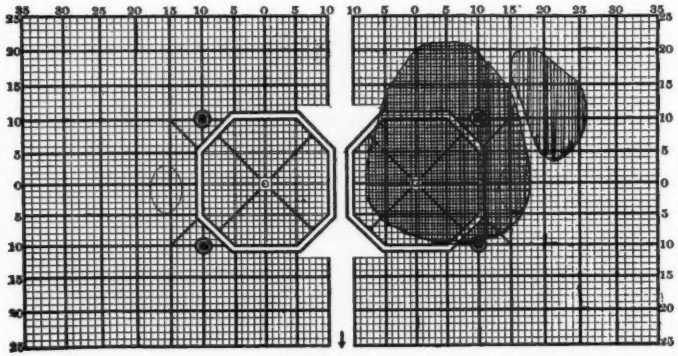

FIG. 1.

Right shows no peripheral contraction, but a large absolute central scotoma with a relative one adjoining up and out. (Fig. 1.) Left normal. She was referred to my asso-

ciate, Dr. W. H. Wimp, for examination of her nose and sinuses and his report is as follows:—"No nasal symptoms. Does not have frequent head colds, but has some mucoid secretion from her nose or nasopharynx early in the morning. Transillumination: Both frontals small but clear; both antra very clear. Both inferior turbinates intumescent. Right middle turbinate missing. No secretion in nose on suction and irrigation. No œdema or thickening in respiratory areas. Both sphenoids probed, posterior walls feel normal. Small amount of caseous material expressed from each tonsil." Dr. J. H. Breyer made a physical examination, including blood, urine and X-ray of teeth. He reported Wassermann negative, Hemaglobin 85%, Reds 3,400,000, Whites 10,000. Albumen and pus present in urine. Teeth negative for abscess. After careful study, by exclusion, we were forced to the diagnosis of retrobulbar neuritis of sinus origin. On October 10, 1922, she was operated by Dr. Wimp at my request. He opened the right posterior ethmoid and the right sphenoid. No evidence of disease was found. On the second day following, October 12, 1922, the vision had improved to $\frac{1}{60}$. October 14, 1922, it was $\frac{6}{15} - 1 + 2$. October 16, 1922, $\frac{6}{15} - 1$. October 20, 1922, $\frac{6}{7} - 2$. October 24, 1922, $\frac{6}{6}$. No scotoma. October 31, 1922, $\frac{6}{6}$. On November 16, 1922, with her correction: O.D.V. $= \frac{6}{6} - 1$.

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AN OPERATION FOR BLEPHAROPTOSIS WITH THE FORMATION OF A FOLD IN THE LID.[1]*

By Dr. ROBERT G. REESE, New York.

(With fifteen illustrations on Text-Plates I.–IV.)

THIS procedure is indicated in congenital ptosis due to either defect in development or entire absence of the levator, and in old cases of acquired ptosis which cannot be cured in any other way, whether the lid is devoid of wrinkles or not. It is also effectual in ptosis myopathica.

I have operated by this method in eighteen cases of congenital ptosis, and in three cases of inveterate acquired ptosis, and the results have been better than those obtained by any of the numerous methods I have employed.

The operation performed as here described corrects the backward pose of the head by permanently elevating the lid and exposing the pupil, thereby restoring the function of the eye. No lagophthalmus is produced during sleep, and the cosmetic effect is materially enhanced by the formation of an extensive fold in the lid.

OPERATION.

The brow and lids are washed with soap and water, and painted with tincture of iodine U.S.P. A general anæsthetic is

[1] Presented before American Ophthalmological Society, Colorado Springs, June, 1923.

* This was the operation referred to in the Archives of Ophthalmology, vol. lii., No. 4, p. 374, as a modified Machek operation. The author would be obliged to any reader who would inform him if he can see any similarity in any step of this operation to the Machek procedure, except the use of the frontalis which is utilized in every ptosis operation except Motais'.

FIG. 1.—AA'. Line of incision through the skin only. Dotted line shows amount of dissection of skin from subcutaneous tissue.

FIG. 2.—AA'. Everted skin.

B. Crescentic area composed of subcutaneous cellular tissue and a portion of the orbicularis. Dotted line shows position of primary incision through the skin.

FIG. 3.—A. Forceps holding up the dissected area of skin and subcutaneous tissue from the convex border of the tarsus and tarso-orbital fascia.

BB'. The two lateral flaps dissected from the tarsus. Sutures passed through their distal ends.

C. 10*mm* of tissue in its normal attachment to the middle of the tarsus.

FIG. 4.—Entrance of the double-edged knife, and its emergence at the external side of the 10*mm* attached tissue with the thread from the external flap in the fenestra. The arrow shows the entrance, direction, and emergence of the knife on the internal side.

FIG. 5.—AA'. Flaps lying on the tarso-orbital fascia.

BB'. Flap sutures emerging through the openings above the brow.

C. Anchor suture with its loop on anterior face of attached tarsal tissue, and its emergence above the crest of the brow.

FIG. 6.—A. Entrance of suture through the opening of skin, and flap. Excess flap to be excised.

B. Tied suture holding the inner flap, approximating the skin.

C. Anchor suture tied over rolled gauze.

DD'. Ajusted skin incision showing extensive fold.

FIG. 1.—AA'. Line of incision through the skin only. Dotted line shows amount of dissection of skin from subcutaneous tissue.

FIG. 2.—AA'. Everted skin.
B. Cresentic area composed of subcutaneous cellular tissue and a portion of the orbicularis. Dotted line shows position of primary incision through the skin.

FIG. 3.—A. Forceps holding up the dissected area of skin and subcutaneous tissue from the convex border of the tarsus and tarso-orbital fascia.
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FIG. 5.—AA'. Flaps lying on the tarso-orbital fascia.
BB'. Flap sutures emerging through the openings above the brow.
C. Another suture with its loop on anterior face of attached tarsal tissue, and its emergence above the crest of the brow.

FIG. 6.—A. Entrance of suture through the opening of skin, and flap. Excess flap to be excised.
B. Tied suture holding the inner flap, approximating the skin.
C. Another suture tied over rolled gauze.
DD'. Aimed skin incision showing extensive loss.

ILLUSTRATING DR. ROBERT G. REESE'S ARTICLE ON "AN OPERATION FOR BLEPHAROPTOSIS WITH THE FORMATION OF A FOLD IN THE LID."

FIG. 1

FIG. 2

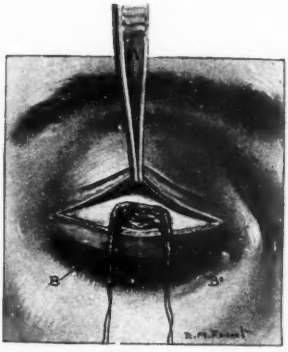


FIG. 3

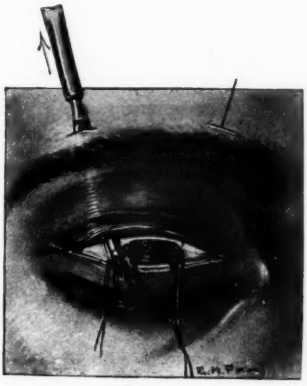


FIG. 4

FIG. 5

FIG. 6

ILLUSTRATING DR. ROBEST G. REESE'S ARTICLE ON "AN OPERATION FOR BLEPHAROPTOSIS WITH THE FORMATION OF A FOLD IN THE LID."

A.

B.

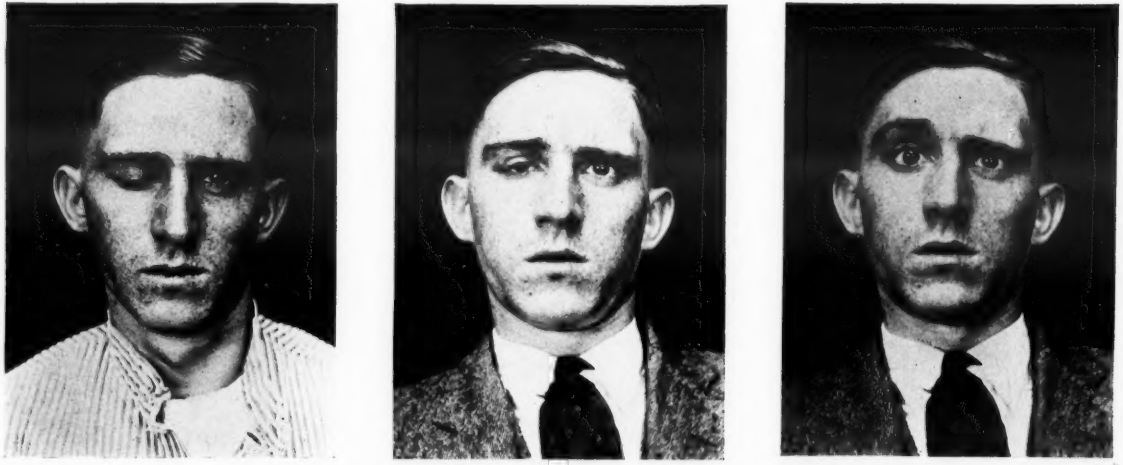
FIG. 7.

A.

B.

FIG. 9.

ILLUSTRATING DR. ROBERT G. REESE'S ARTICLE ON "AN OPERATION FOR BLEPHAROPTOSIS WITH THE FORMATION OF A FOLD IN THE LID."



A. B. C.

FIG. 8

ILLUSTRATING DR. ROBERT G. REESE'S ARTICLE ON "AN OPERATION FOR BLEPHAROPTOSIS WITH THE FORMATION OF A FOLD IN THE LID."

A. B.

FIG. 10

seldom necessary, so that the lines of the incisions are injected subcutaneously with a solution of novocain 2%, with 5 drops of solution of adrenalin 1:1000 added to each *cc.*

After ten minutes insert a Jäger horn plate beneath the upper lid, and make a curvilinear incision through the skin only, from one extremity of the lid to the other, so that the center of the curve will be 6*mm* from the lid margin, and the extremities of the incision 4*mm* from the lid margin. (Fig 1.) The upper and lower portions of the skin adjacent to the incision are dissected from the subcutaneous tissue 4*mm* above and 2*mm* below. (Fig. 1.)

An incision is then made beneath the skin and 2*mm* below the lower margin of the curvilinear incision down to the tarsus, and parallel to the lid margin from one extremity of the lid to the other. A second incision is made beneath the upper skin flap and joining the extremities of the first incision, so that the central portion of this crescentic area lying between these two incisions will be 6*mm* vertically. (Fig. 2.)

The sides of this crescentic area are dissected from the tarsus, leaving 10*mm* of the central area in its normal attachment to the tarsus, thereby forming two lateral flaps. (Fig. 3.) The skin and subcutaneous tissue above this central portion of the crescentic area are dissected from the underlying tarsus and tarso-orbital fascia 6*mm* vertically and 15*mm* horizontally, in order to allow the flaps and the 10*mm* attached portion to be drawn up beneath them, so that they will become adherent to the tarso-orbital fascia and convex border of the tarsus. (Fig. 3.)

Enter a double-edged knife (Fig. 4) just above and external to the crest of the eyebrows and push it downward, hugging the tarso-orbital fascia, so that its point emerges at the side of the 10*mm* attached central portion of tissue, at an angle of 25°. (Fig. 4.)

A suture is passed through the end of the flap, and the ends threaded in the fenestra situated near the point of the double-edged knife (Fig. 4), so that by withdrawing the knife the flap is pulled up and the thread protrudes through the opening above the brow. (Fig. 5.) An analogous procedure is done on the inner side, above the eyebrows, with the same knife, avoiding the supra-orbital nerve. (Fig. 5.) One needle of a

double-armed, twisted silk thread is entered *2mm* below the upper central border of the *10mm* portion of attached tarsal tissue, as an anchor suture (Fig. 5), and passed upward beneath the subcutaneous tissue, emerging above the crest of the eyebrows; the other needle is entered the same way, *3mm* horizontally from the entrance of the first needle, and carried upward in the same manner, forming a loop on the anterior surface of the tarsal attached subcutaneous tissue. (Fig. 5.) The thread is tied over a piece of plain rolled gauze, and drawn up sufficiently to produce a marked lagophthalmus. (Fig. 6.)

The lateral flaps are drawn through the openings above the brow with the threads, so that they lie smoothly on the tarso-orbital fascia and without producing any traction on the lid. (Fig. 6.) A suture is then passed from above downward through the upper edge of the skin opening, protruding flap, and lower edge of the skin opening, and the excess of flap excised. (Fig. 6.) The suture is tied after the flap is allowed to recede below the skin surface, in order that it may become adherent to the subcutaneous surface, and is not interposed between the margins of the incision, thus avoiding a puckered scar. The curvilinear opening in the lid is adjusted, and no sutures are used. (Fig. 6.) Borated vaseline is applied abundantly for protection and to prevent dessication of the cornea. Rubber tissue and plain gauze are used and a bandage is applied. It is advisable to inspect the eye after 24 hours and use borated vaseline as a protection at each daily dressing.

There should be considerable lagophthalmus after the operation which will disappear in a few days.

In the after treatment it is important that the sutures are not removed too early, otherwise the elevation of the lid may not be sufficient, and, moreover, the union between the tarso-orbital fascia and the flaps would not be firm enough; consequently the sutures are removed after seven days.

The bandage may be left off after four days but the sutures should then be covered with adhesive strips.

The contraction of the frontalis pulls on the submerged flaps, raising the tarso-orbital fascia and tarsus, simulating the action of the levator, and the lid does not become everted in its elevation, but hugs the globe normally. The displacement of a portion of the orbicularis does not impair its normal func-

tion of closing the lid, so that lagophthalmus during sleep is prevented, and the reflex palpebral closure is preserved.

If the skin of the lid is redundant, which is not usually the case, a segment may be removed, although an excess of skin improves the cosmetic result rather than otherwise.

In eight of my first operations dermic flaps were used, and in each instance a sebaceous cyst formed along their course, notwithstanding the epithelial surface was scarified and scraped, a procedure advocated and practiced by me fifteen years ago when the Tansley-Hunt was my operation of choice.

I am adding the histories of four cases, three of which were operated on by other operators.

CASE I.—(Fig. 7.)—Operated on by Dr. C. E. O'Connor of Kingston, Ontario, who kindly supplied me with the photographs and the following history:

Male. Age 33. Congenital ptosis of both eyes. Had Gillet de Grandmont operation on both eyes ten years ago, and the result was as shown in Fig. 7.

A—August, 1921, Reese ptosis operation on both eyes.

B—Two months after operation.

CASE II. (Fig. 8.)—Operated on by Dr. Ben Witt Key, of New York, to whom I am indebted for the photographs and the following history:

Male. Age 35. Monocular congenital ptosis and paralysis of the right superior rectus. Reese ptosis operation December, 1922.

A—Before operation.

B—After operation. Frontalis in repose.

C—Frontalis in action.

CASE III. (Fig. 9.)—Operated on by Dr. Ben Witt Key, who kindly supplied the photographs and the following history:

Male. Age 36. Bilateral congenital ptosis.

A—Before operation.

B—Three weeks after Reese's ptosis operation.

CASE IV. (Fig. 10.)—Female. Age 16. This was the first case I operated on. Bilateral congenital ptosis, and paralysis of the inferior rectus with convergent strabismus of the right eye.

V.R.E. $\frac{20}{200}$ unimproved, amblyopia ex anopsia. V.L.E. $\frac{20}{15}$.

First operation, December, 1916. Dermic flaps were used.

Sebaceous cysts formed along these submerged flaps in the course of six months, and were dissected out.

January, 1917, Reese resection of the external rectus and tenotomy of the internal rectus was performed.

December, 1918, ptosis operation as here described, performed on both eyes.

A—After the first operation, as no photograph was taken before the operation.

B—Five years after the second operation.

DUCT CONSERVATION IN LACRIMAL ABSCESS.

By A. E. EWING, M.D., St. Louis, Mo.

(With eight illustrations on Text-Plate V. and one in the text.)

THE crudeness of our knowledge with regard to the anatomy of the lacrimal duct is doubtless the reason why the probe has acquired its unpleasant reputation as an instrument of torture both with patients and physicians in the treatment of lacrimal abscess and other lacrimal obstructions. Because of the difficulty in dissecting the duct it is seldom seen and studied in its natural state during our student period. After leaving the anatomical room few of us return later for closer investigation.

Our present means of decalcification has enabled us to clear away the haze in the management of this class of cases so far as concerns the anatomy, as is demonstrated in the accompanying illustrations, natural in size, from photographs of decalcified specimens.

There are two cross-sections of the duct (Figs. 1 and 2) which show the relation of the soft tissue to the bony wall; two antero-posterior sections seen from the nasal side which show the size of the lacrimal sac and the entrance of the canaliculi into the sac. In one of these (Fig. 3) the valve of Hasner is a membrane surrounding a large round opening into the nasal fossa at the lower margin of the inferior turbinate articulation. In the other (Fig. 4), this valve is anatomically perfect, as is demonstrated by the flexible probe which lies in the duct and passes through the valve from the articulation of the inferior turbinate to the normal depression in the outer nasal wall, which is the exit of the duct. Also in this figure the entrance of the canaculi into the sac is shown by a probe which

enters through the upper canaliculus. There are five lateral sections which show the duct from the front. One of these, to the left in Fig. 5, has a large round opening into the nasal fossa. The others have more or less perfect valves of various lengths. The one containing the probe illustrates the valve held away from the nasal wall by the probe (Fig. 8). In Fig. 5, left eye, the size and course of the canaliculi from the puncta to the sac are clearly depicted.

As the illustrations represent the actual size and length of the lacrimal passage they are guides in the selection of probes, the majority of which are too long and too straight and those ordinarily employed are too large. The large probe injures the tissues of the passage from the punctum to the valve of Hasner. The probe that is straight or in line with the antero-posterior plane of the duct will cause pain by pressing on the outer wall of the nose. It may even crush through this wall into the antrum. The long probe is a cruel instrument because it punches into the floor of the nose and causes excruciating pain. Probes should be so constructed that their length from the finger plate will not permit them to touch the floor of the nose. When in use the tips should be curved toward the median line, sometimes sharply as indicated in Fig. 9, C2, taken from two cases in each of which this curve was necessary in order to enter the nasal fossa.

A glance at the five illustrations seen from the front will demonstrate the correctness of this observation. Even the very straight duct of Fig. 7, a straight probe would injure the nasal wall after passing the articulation of the turbinate if it were pushed to the floor of the nose; in the crooked duct of Fig. 6, it would pass directly into the antrum. This is further demonstrated by the curve of the flexible probe in the duct of Fig. 8. Usually a slight curve nasalward in the metal probes is all that is necessary as shown in Fig. 9, C1. The flexible probe will follow the natural channel. As seen from the side the probe should be straight instead of bent forward, which is the usual custom. A probe bent forward will become entangled at the entrance of the sac into the bony duct or at the firm articulation of the lower turbinate and maxillary bones.

The sizes of probes to be used in each case should be governed by their ease of passage through the canaliculus. Usu-

Description of the Illustrations of the Lacrimal Passages.

Fig. 1.—Horizontal sections through the lacrimal sac and duct. In the upper right section one looks upward into the sac. In the other sections one is looking downward into the sac and duct. In the two upper sections the entrance of the canaliculi into the sac is shown. The sections illustrate the relation of the mucous and submucous or soft tissues of the duct to the bony wall, the white ring being the soft tissue within the bony wall.

Fig. 2.—Horizontal sections through the lacrimal sac, the duct, and the valve of Hasner (V. H.). They illustrate the relation of the soft tissue to the bony wall, the outer dotted ring being the bony wall.

Fig. 3.—A lateral view of the right lacrimal sac and duct from the nasal side. The anterior boundary of the sac is indicated by the dotted line. In the upper left portion of the sac the entrance of the canaliculi is shown by the short nearly vertical dark line. The exit of the duct into the nasal fossa is a large round opening in the mucous membrane—an imperfect valve of Hasner (V.H.). Just below this to the left is an opening into the antrum and to the right a cross section of the lower turbinate. This was a large duct with a thin mucous and submucous lining.

Fig. 4.—A lateral view of the left lacrimal sac and duct seen from the nasal side. It illustrates the lacrimal sac, the entrance of the canaliculi into the sac (indicated by a probe passed through the upper canaliculus), the soft tissues of the duct, and an anatomically perfect valve of Hasner. The valve is shown by the flexible probe that lies in the duct and passes through the valve from the articulation of the inferior turbinate to the normal depression in the outer nasal wall, which is the exit of the duct.

Fig. 5.—A front view of two lacrimal sacs and ducts. The right duct has a large round exit into the nasal fossa in a fold of mucous membrane. The valve in the left duct was normal but was cut away in making the section. The vascularity of the soft tissues within the duct is here illustrated. The size and direction of the left canaliculi and their entrances into the sac are very well depicted.

Fig. 6.—A front view of the lacrimal sac and duct. The tissues in front of the canaliculi were cut away in order that their direction might be outlined. The soft tissues within the duct and the possibility of a crooked duct are well indicated. The valve of Hasner extends far down on the nasal wall.

Fig. 7.—A front view of the lacrimal sac and duct. It shows a straight duct which is well supplied with mucous and submucous tissue.

Fig. 8.—A front view of the lacrimal sac and duct. A flexible bulb probe lies in the sac and duct. The nasal end of the probe holds the valve of Hasner (V.H.) away from the nasal wall.

It should be noted that the nasal end of this probe curves to the median line. Were the probe straight it would enter the antrum, yet this is a reasonably straight duct.

DESCRIPTION OF THE ILLUSTRATIONS OF THE LACRIMAL PASSAGES.

FIG. 1.—Horizontal sections through the lacrimal sac and duct. In the upper right section one looks upward into the sac. In the other sections one is looking downward into the sac and duct. In the two upper sections the entrance of the canaliculi into the sac is shown. The sections illustrate the relation of the mucous and submucous or soft tissues of the duct to the bony wall, the white ring being the soft tissue within the bony wall.

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FIG. 4.—A lateral view of the left lacrimal sac and duct seen from the nasal side. It illustrates the lacrimal sac, the entrance of the canaliculi into the sac (indicated by a probe passed through the upper one looking), the soft tissues of the duct, and an anatomically perfect valve of Hasner. The valve is shown by the flexible probe that lies in the duct and passes through the valve from the articulation of the inferior turbinate to the normal depression in the part of nasal wall, which is the exit of the duct.

FIG. 5.—A front view of two lacrimal sacs and ducts. The right duct has a large round exit into the nasal fossa in a fold of mucous membrane. The valve in the left duct was normal, but was cut away in making the section. The asymmetry of the soft tissues within the duct is here illustrated. The size and direction of the left canaliculi and their entrances into the sac are very well depicted.

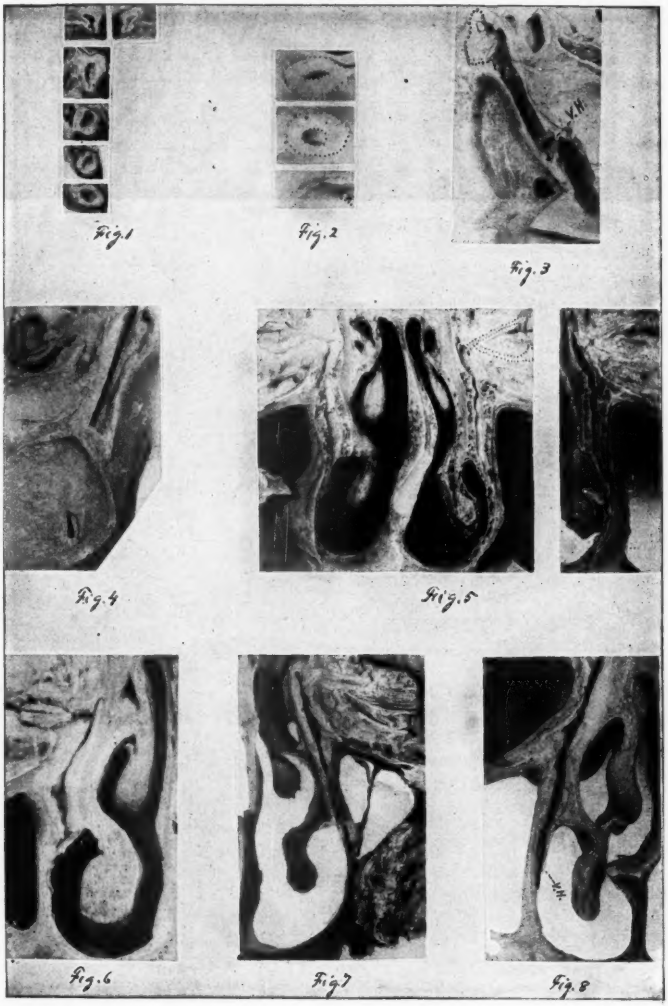
FIG. 6.—A front view of the lacrimal sac and duct. The tissues in front of the canaliculi were cut away in order that their direction might be outlined. The soft tissues within the duct and the possibility of a crooked duct are well illustrated. The valve of Hasner extends far down on the nasal wall.

FIG. 7.—A front view of the lacrimal sac and duct. It shows a straight duct which is well supplied with mucous and submucous tissue.

FIG. 8.—A front view of the lacrimal sac and duct. A flexible bulb probe is in the sac and duct. The end of the probe holds the valve of Hasner (V. H.) away from the nasal wall.

It should be noted that the nasal end of this probe curves to the median line. Were the probe straight it would open the antrum, yet this is a remarkably straight duct.

ILLUSTRATING DR. A. E. EWING'S ARTICLE ON "DUCT CONSERVATION IN LACRIMAL ABSCESS."



The lacrimal passages, eight illustrations, natural size.
Figs. 1 and 2 are cross sections, 3 and 4 are antero-posterior sections, 5, 6, 7, and 8 are lateral sections.

ally a probe which is one millimeter or one and two tenths of a millimeter in diameter will pass without force. On this basis probes should be constructed with a gradation in sizes of one

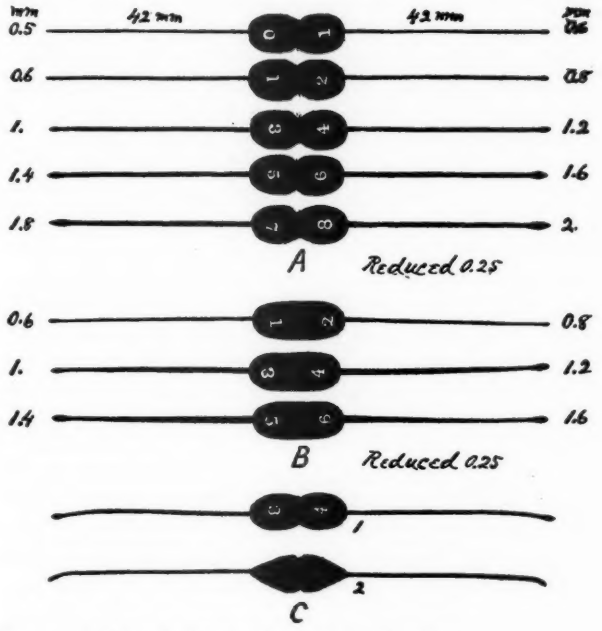


FIG. 9.—A. Silver bulbous probes tipped with solder.
B. Whalebone flexible probes with bulbous tips.
C. 1. The nasalward curves usually required in order to safely pass through the majority of lacrimal ducts.
2. Probes showing the nasalward curves that were necessary in two cases in order to open obstinate strictures at the lower end of the ducts.

fifth of a millimeter in diameter using the one millimeter size as the standard. The accompanying probes (Fig. 9,A) are constructed with this gradation, the 0 being any size less than 0.5*mm*, as these sizes readily pass the puncta and canaliculi. They can be constructed to any size desired by using for the shaft hard-drawn silver, gold, or steel (piano) wire, and for bulb probes by tipping the points with solder. In order to

protect against their being pushed into the floor of the nose their length from the finger plate should not exceed 45*mm*. In several ducts that have been measured, the distances from the puncta to the floor of the nose have varied from 42 to 45*mm*, the majority being of the shorter length. For this reason I have assumed 42*mm* as being a harmless length for the probes, and yet a length that will extend below an anatomically perfect valve of Hasner when the probe has been entered from either punctum.

The bulb probes suggested by Travers, 1824, and in 1867 adopted by Dr. Williams, are superior to those of Bowman because they more readily locate strictures and they injure the lacrimal passage only in the region of the strictures.

Other valuable probes are those made from whalebone, either of the Bowman or the Travers type as they may be obtained in any size and if too stiff they are easily softened by boiling. In the bulbous form they are especially useful for diagnosis, little or no pain being caused by their introduction. Frequently they are curative without resorting to metal probes. Their length may be gaged by substituting for the metal finger plate a finger hold made from the rubber tire mending substance known as "tire putty" or fixing a finger plate to the probe by means of the putty. This is easily applied and after it is fixed it resists both alcohol and boiling. Such probes are shown in Fig. 9, B.

Previous to dilating the punctum or dividing it for the entrance of a probe there should always be thorough anæsthesia. This may be accomplished in ten or fifteen minutes with cocain or holocain or both combined. The upper canaliculus should always be the choice, as it is shorter and the arc of the curve for entering the bony duct is only about half the arc when the lower canaliculus is chosen. Also the lower canaliculus is the chief drainage canal, and should be carefully protected from injury. If the incision is resorted to it should extend along the canaliculus for a length not greater than 3*mm*. Such an incision is harmless as it does not interfere with the valvular function of the lacrimal sac. It is best performed with a curved Weber knife, the edge of the knife being directed backward in order to conceal the incision beneath the lid margin.

Following the dilating or the incision, the sac and the duct

should be anæsthetized by means of cocain 5% injected with a syringe. For this injection I prefer the Shahan syringe because it is so readily sensitive to pressure. At the same time that the duct is anæsthetized the nasal region of the affected side should be anæsthetized with a solution of 5% cocain in an adrenalin solution of 1:4000 by means of a swab in order to anæsthetize the nasal end of the duct and also shrink the inferior turbinate so that the end of the probe may be seen on the outer wall of the nose after it has been successfully passed.

In its manipulation the probe should be introduced horizontally in the manner usually described, until the nasal wall is reached, the finger end should then be raised to a vertical position and the probe end should be guided into the duct by pressing the forefinger over the sac. Usually it is best to massage strictures. This is easily accomplished with a bulb probe. The location and the number of strictures should be noted in the record for future reference and comparison.

The nasal tip of the probe should always be observed with a head mirror as a guard against provoking a false passage. Care should be exercised not to do any syringing immediately after the probe has been passed, as the fluid might extravasate into the soft tissues of the orbit and cause an unfortunate swelling. It is best to wait for the syringing until the next day or sometimes two days. However, the duct and the sac should be kept clean by means of a mild collyrium and pressure, and if the canaliculus has been incised, the incision should be reopened daily with a small probe for a week or ten days until epithelium has covered the edges of the wound.

Syringing should be resorted to when pus or mucus is present. For this purpose the solutions that give greatest relief are mercurochrome 2%, zinc sulphate 0.5 solution and the silver salts. The latter should not be used frequently for fear of argyrosis. Anæsthesia should always precede the syringe.

The employment of probes is often discouraging because they frequently do not accomplish an immediate result. As a rule, however, there is an immediate improvement, especially when it is taken into account that these ducts are chronically poisoned. By eliminating the poison the swelling along the duct will subside and new epithelium will partially or completely cover the injury of the probe in the stricture. If par-

tially, a second probing should be resorted to in six or eight weeks, not earlier than this, as time between probings should be given for chronic swelling to subside and for new epithelium to be established. As is shown by the size of the puncta, any small opening that can be obtained will establish drainage. By careful persistent treatment this may be accomplished in any case except displacement through injury or where there is malignancy.

For my anatomical material I am indebted to Dr. Robert J. Terry, Professor of Anatomy at Washington University, for two of the specimens, and for the remainder to Dr. Greenfield Sluder, Clinical Professor of Laryngology and Rhinology in Washington University, who has always made a point of having a large decalcified supply on hand for the use of his students and assistants. For selecting the material I wish to acknowledge the assistance of Dr. W. B. Hoover and Dr. M. F. Weymann and also the aid of the latter in sectioning and mounting the specimens.

AN ANALYSIS OF THE COMPENSATION PROBLEM AS IT RELATES TO OCULAR INJURY.[1]

By Dr. ALBERT C. SNELL, Rochester, N. Y.

(With three figures in the text.)

THE problem of appraising compensation for ocular injury, or for injury to any other part of the human organism, is a complex and complicated one, but its solution does not present insurmountable difficulties when a careful and thorough analysis is made of the various elements which enter into the problem. In order to determine the nature of any complex organism the logical method of procedure is to resolve the complex whole into the various elements or factors which compose the composite whole, and then to find out the relationship of such elements or factors to one another. This method of analysis is peculiarly apposite in determining the power of any complex machine or organism to do work. I believe that such an analysis of the compensation problem will reveal fundamental principles which may be clearly understood, and which may be formulated into scientific truths which will make clear the proper solution of this problem. We shall attempt such an analysis of the problem of computing compensation for ocular injury. That this problem needs clarification is evident from the fact that many different schemes have been presented during the last few years, and that these give us a wide diversity of conclusions.

The compensation problem resolves itself into five distinct phases, each of which has an important relationship to a proper understanding of its solution. We shall discuss the problem from these five aspects:

[1] Read before the Buffalo Academy of Medicine, January, 1923.

1. The basis on which compensation rests, which is fundamentally a loss of earning ability.

2. The philosophical and mathematical relationship of essential elements which compose any complex act or function such as earning ability or the visual act.

3. The relationship of visual function to general bodily function.

4. The numerical measurement of the essential elements of full visual function.

5. The legal concept or scope of present compensation laws and the relationship of some proposed methods for computing compensation to these laws and to accepted economic principles.

I. The Basis of Compensation.

The first step in the analysis is the determination of the basis on which compensation rests. This basis can be easily found by a careful study of the statutes according to which compensation is computed. From such a study we find that workmen's compensation is an attempt to award damages for a loss sustained by the human organism, due to industrial injury or disease. This loss is expressed by such terms as "incapacity," "disability," "loss of use," and other terms of similar meaning. In the New York State Workmen's Compensation Law we find that compensation is based on the "loss of use of a member," or a loss to "wage-earning capacity"; that awards are made for "decreased earning power," and disability is defined thus: "'Disability' means the state of being disabled from earning full wages at the work at which the employee was last employed." From these expressions, and from the language used in nearly all compensation statutes, the deduction is clearly brought forth that fundamentally the "loss" for which indemnity or recompense is to be made is that loss of use of a member or part which results in a decrease in the ***ability to earn***.

This conception of the problem involves more than the consideration of loss to function only, although loss of function is always primary. It is quite apparent that "wage-earning capacity" or earning ability is not synonymous with working ability, since a person may have considerable working ability

and at the same time have no earning ability. He may be able to work at something, but he may not be able to get any work to do. For example, a blind locomotive engineer whose other functions are all normal would have power to work, but as an engineer his earning ability would be nil. Thus we see that the compensation statutes have based compensation correctly on loss to earning ability.

It is obvious that "earning ability" is a complex quantity; therefore, in order to understand it, let us analyze this complex quantity by finding out what are the various elements or factors which compose it, and determine the relation of such elements to one another. The elements which enter into "earning ability" have been carefully considered by many students of physical economics and they agree that there are three indispensable factors. These are functional ability, technical ability, and competitive ability. It does not need an extended argument to show that the ability of anyone to earn a living depends primarily on the degree of efficiency at which the various parts or organs of the body are functionating; if the human organism is not functionating it has no power to earn anything. Second, it is self-evident that no one can accomplish any work or earn anything unless he has some knowledge of how the work is to be done, no matter how simple the work may be; even the laborer with pick and shovel must have some knowledge of how to use his tools. Third, even when a person has perfect bodily function and the necessary technical knowledge which would make it possible for him to do work, in an economic sense he could not accomplish anything or earn anything if he could not get employment. Therefore, anything which interferes with one's ability to compete in the productive field or in the labor market interferes with one's earning ability. Thus is it demonstrated that each of these three elements is an indispensable factor, since with any one of these elements absent, the earning ability is nil. Not only is each of these factors of the earning ability indispensable to the full earning power, but it is also apparent that there is a co-ordinate, reciprocal relationship with one another. The work accomplished by the functional activity is directed by and is dependent on the technical knowledge, and the ability to do work is dependent on the ability to get work to do.

II. The Mathematical Relation of Essential Elements.

Having determined the nature of these three elements and their relationship to one another as they relate to the full earning ability, it is next necessary to express this abstract power, "earning ability," in numerical quantities. Since the problem of determining compensation is a mathematical one we obviously must use the science of numbers for its solution. Then, in what way can one express numerically the relationship of these three elements, which shall satisfy the premise that each factor is indispensable, and that each has a co-ordinating, reciprocal action in making up the full normal earning ability? In general two suppositions have been advanced to express the elements of the earning ability in numerical terms.

I.—The first supposition divides the whole quantity into fractions.

II.—The second supposition resolves the whole quantity into mathematical factors.

It is obvious that a number or quantity either may be divided into fractional parts which when added together make the whole, or that it may be resolved into factors which when multiplied together make the whole. Let us proceed by logical analysis and deduction to determine which of these methods will satisfy the premise laid down above.

For simplicity, let E represent full earning ability, let F represent functional ability, let T represent technical ability, and let C represent competitive ability. Taking up the first supposition, we might assume that the three elements which make up full earning ability, F, T, and C, have equal fractional values; then the value of each would be $\frac{1}{3}$, and the equation to express the whole would be, $\frac{1}{3}(F) + \frac{1}{3}(T) + \frac{1}{3}(C) = 1(E)$. Such a supposition denies that each of these three elements is indispensable and that they have a co-ordinate, interdependent relation. This is shown in this purely mathematical formula when one element is absent, $0(F) + \frac{1}{3}(T) + \frac{1}{3}(C) = \frac{2}{3}(E)$. This equation, assuming that one element may be absent, says that it may be dispensed with. But such a conclusion is directly opposed to our premise that each element is *indispensable.* This supposition also leads to an absurd conclusion. Assum-

ing that the first element—that of function—was absent, by adding the remaining two elements, we obtain the absurd conclusion that earning ability is still ⅔ efficient, whereas with all bodily function lost a man is of course dead. The same conclusion must be reached in considering the relation of the other two elements. Thus it is demonstrated that indispensable, co-ordinating elements which compose such a complex force as earning ability, cannot be added together in attempting to assign to them a numerical value. It is evident that this supposition does not satisfy the premise and must be discarded.

Taking up a consideration of the second supposition—that of regarding each essential co-ordinating element as a mathematical factor—we may at first assume that each element of full earning ability has equal value. Thus each must be regarded as a unit, because the three equal factors of one are one. Then we may express the full earning ability, using three factors, by the formula $1(F) \times 1(T) \times 1(C) = 1(E)$ or full earning ability. This method completely satisfies the condition laid down in the premise since the product is 0 when any factor of the entire quantity becomes 0. For example, $0 \times 1 \times 1 = 0$. Thus the loss of any one of the three elements of the earning ability nullifies completely the earning ability, which fact makes each element an essential—indispensable—element. The complete formula for expressing this truth is that of Magnus which is $E = F \times T \times C$. This is the only method by which the full earning ability may be expressed by a numerical quantity or formula which takes into consideration the fact that there are these three indispensable, co-ordinating elements which compose the earning ability. This conclusion was first reached by Magnus, and since the publication of Magnus on this subject all thoughtful students of visual economics have accepted this conclusion of his as correct in principle.

A philosophical consideration of any complex functionating mechanical or physical organism will reveal the fact that the relation of those essential and co-ordinating elements which unite to produce action or function is governed by the *law of essential factors*. This scientific law is:—*Essential co-ordinating factors of any complex organism stand in the relationship to one another as do the factors of a product.* The discussion above

in regard to this relationship of the essential factors which compose full earning ability shows that these elements are governed by this law. The writer[1] has recently given other concrete examples illustrating the operation of the law of essential factors to which the reader who may desire further demonstration of the operation of this law is referred. Later in this paper it is shown that visual function is governed by this law and also that the essential elements of vision are likewise governed by the operation of this law.

Having demonstrated above that the elements which enter into the complete earning ability are indispensable and therefore stand logically and satisfactorily in the relation to one another as do the factors of a product, *and not as elements* of a sum, we may proceed to consider how each of these factors influences the product under all practical circumstances. As Dr. Holt[2] has clearly pointed out there are just three things which can possibly happen to the earning ability from injury or disease, namely: (1) no loss to it, (2) partial loss to it, or (3) total loss to it. Evidently, when there has been no loss to the earning ability there will be no compensation problem to solve. Also, when there has been a total loss to earning ability there will be no compensation problem, because nearly all compensation laws specify definite rates and amounts of awards for the total loss of earning ability or for total incapacity. Thus from a practical point of view we need to consider only the cases of partial loss to earning ability.

In order to determine the amount of damage to earning ability when there has been a partial loss to it from injury or disease, we must consider to what extent each individual factor may be damaged, how each may influence the product, and how the numerical value of each may be determined and expressed. We shall find that the numerical value for each individual factor which has sustained a partial loss must necessarily be a fraction of the whole. Therefore, the fraction which expresses the remaining functional value of such an impaired element is the coefficient of this element. For example, if

[1] Snell, "Estimating Compensation for Ocular Injury," *Eye, Ear, Nose, and Throat Monthly*, Aug., 1922.

[2] Holt, "Visual Economics," *American Encyclopedia of Ophthalmology*, vol. xviii., and *Trans. American Ophthalmological Society*, vol. xviii., p. 298.

function is impaired 50%, the coefficient of F is .50, expressed as .50(F), etc.

We shall take up first the consideration of the manner in which factor T, the technical ability, may be damaged. In view of the fact that this factor is never changed except in brain injury or disease, as has been pointed out by Magnus and by Holt, it may be left out of the computation except in the consideration of brain injuries where it is included under functional brain disturbances. It is apparent that a person's technical knowledge is not affected in any way by damage to other bodily functions. For example, any damage to hands or to feet or to ocular function does not alter or change the knowledge one may possess. If this factor were left in the formula it would always remain as the unit (1),—its coefficient would always be (1),—and thus would not alter in any way the final product, since any number or quantity multiplied by (1) is not changed in value. Thus the formula is simplified and we have only to consider the damage to the other two factors.

The two remaining factors, functional ability and competitive ability, which are essential to full earning ability, are so inter-related that we must consider first their co-ordinate action before attempting to determine the numerical value of each. We find that damage to function is necessarily a primal step before there can be any loss to earning ability, and that the competitive factor is entirely dependent on the status of efficiency of the former; for it must be self-evident that the competitive ability cannot be damaged by itself, but can only be influenced in conjunction with some degree of loss to function. One cannot conceive of a direct injury to competitive ability alone and one cannot conceive of any degree of damage to earning ability without some degree of damage to function. Thus functional ability is the keystone to the solution of our entire problem and every degree of damage primarily must be based on damage or loss to functional efficiency; whereas competitive ability is a secondary factor and is entirely dependent on the status of the functional efficiency.

The discussion thus far and the principles which this discussion have established are applicable to injury to any organ or function, but I shall throughout the rest of this paper direct

your attention entirely to ocular disturbances. We must first determine the relation of ocular function to total bodily function in order to establish a numerical measure for visual function. Is visual working ability synonymous with total working ability?

III. Relation of Ocular Function to Earning Ability.

At the very beginning of this phase of our subject the consideration of the relationship of ocular function to the total complex function of the entire human system must be established, because an important step in the solution of our problem depends on our conception of this relationship. On this point there arises a divergence of opinion between two of our leading authorities on visual economics. Magnus[1] holds to the view that visual earning ability is synonymous with earning ability as a whole. This view is clearly set forth in *Visual Economics*, from which I quote the following: "In this work we start with the supposition that the earning ability for any gainful vocation requiring eyesight is practically synonymous with the visual earning ability. . . . It is self-evident that a totally blind person is absolutely incompetent to work at any trade or in any profession which demands eyesight, and that the vast majority of blind people are not only incapable of earning anything, but are a charge upon their families and upon the community. . . . There can be no doubt that the possession of good eyesight is the prime factor for full earning ability for the greater majority of gainful human vocations. . . . We consider that with laborers or artisans, in fact any of the so-called working classes, who may by accident acquire some defect of eyesight, ranging from partial to complete blindness in one or both eyes, the effect of this visual defect upon the earning ability will be identical with the percentage of the loss of eyesight or visual earning ability." Holt[2], however, holds a different view. He states that the functional ability of the eyes is only "an important part" of the functional ability of the body. Holt estimates by empirical

[1] *Visual Economics*, Magnus and Wurdemann, p. 11.

[2] *American Encyclopedia of Ophthalmology*, vol. xviii., and *Trans. Am. Ophthalm. Society*, 1920.

methods that the numerical relative value of one eye to that of total bodily function is a fraction, being .18 or 18%, and that the relative value of both eyes to total bodily function is .36 or 36%; or in other words there is a remaining functional efficiency of 64% when a person is entirely blind. In the formula $F \times C = E$, this suppostion makes the coefficient of F a fraction (.64) when there is total loss of visual function in both eyes.

In the opinion of the writer this assumption of Dr. Holt may be criticized from three points of view.

I.—It is not in harmony with economic fact.

II.—It is not consistent with the law of essential factors.

III.—It is not in agreement with general and accepted authoritative opinion, nor with the economic concept of most of the statutes on compensation.

In regard to the first point, we find that every economic consideration leads one to the conclusion that blind persons, especially those who have acquired blindness after adult life and who are working for a living, have sustained a permanent total disability. Hence blindness is a total working incapacity. Therefore, one cannot assume that the total loss of visual function is a fraction of total bodily function; nor can one consider the coefficient of F in the formula $F \times C = E$ as .64, which means that a person who is totally blind is 64 per cent. functionally efficient. Dr. Holt also shows that the loss of both eyes is just twice as serious as the loss of one eye. This position is not consistent with economic fact. It is generally agreed that the loss of one eye is only $\frac{1}{3}$ as serious as the loss of both eyes, or conversely, that the loss of both eyes is five times more serious than the loss of one eye. In regard to the second point, if vision is an indispensable factor to working ability it cannot be regarded as having a fractional value of 36% of total bodily function. By the law of essential factors, the loss of any indispensable (essential) factor of any complex functionating organism necessarily causes a total functional loss to that organism. This point has been presented by the writer in previous discussions[1] of this subject. In regard to the third

[1] *Eye, Ear, Nose and Throat Monthly*, Aug., 1922, p. 315; *Trans. of Section on Ophthal., A. M. A.*, 1922, pp. 10 and 11.

point, we find that various forms of health and accident insurance regard the total loss of both eyes or of vision as a total economic disability. The New York State Workmen's Compensation Law states, "Loss of both hands, or both arms, or both feet, or both legs, or both eyes, . . . shall constitute permanent total disability." In addition to New York the statutes of California, Colorado, Delaware, Idaho, Missouri,

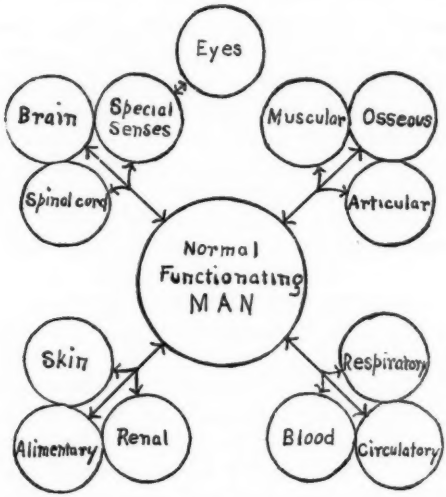


FIG. 1. Illustrating the co-ordinate relation of the essential functioning systems of the human organisms.

Ohio, Oregon, Utah, and West Virginia specifically provide that the total loss of vision in both eyes shall entitle such claimant to the maximum compensation for life. Other states pay an amount for the total loss of vision which is equal to that for any other form of disability. Thus the statutes of nearly all of our states recognize that blindness causes total working incapacity or that its loss is as serious as that of any other form of disability.

The accompanying graph clearly depicts that untrue and absurd conclusions will be formulated in regard to functional efficiency if we attempt to divide the entire human system

into different independent functional systems and assign to each an independent partial or fractional value. From the point of view of functional efficiency or of usefulness of any part or organ it is clearly apparent that when any one of the four great systems of the human organism is lost, there is total loss of function, or when any one of the twelve minor but essential parts is lost there is a total loss of function. For example, it must be self-evident that the human body cannot functionate without blood, or without a heart, or without the respiratory system or without any of the other systems. Therefore one cannot assign to each of these major systems a functional value of $\frac{1}{4}$, nor a fractional value of $\frac{1}{12}$ to any of the minor but essential functional parts, since the loss of any one of these twelve essential parts would cause a total loss of all function—death. Thus it is plain that each system must be complete in itself in order that the entire human system may functionate 100 per cent. efficiently and it is also plain that each of these systems has an interdependent, co-ordinating, functional relation so that they are governed by the law of essential factors. Therefore the coefficient of each of these systems must be 1 for full normal function, and 0 for total loss of any. (The coefficient for total loss to any of these systems cannot be a fraction.) Although the loss of both eyes does not cause death, from the point of view of functional efficiency or the ability to do work, the total loss of vision does cause a total working disability to anyone whose work depends on sight. Therefore vision should also be regarded as an essential factor of working ability. The ultimate numerical value of any function can only be measured by the standard of usefulness in accomplishing work just as we would measure any other physical force. The loss of those members of the body which causes a total incapacity to do work necessarily causes at the same time a total loss of functional usefulness. Economists have commonly regarded the loss of both eyes, or of both hands, or of both legs as a total loss of the working ability. If vision is an indispensable element of "working ability" it is synonymous with full working ability, and normal vision must be regarded as an integral unit factor, and total loss of vision must be regarded as 0 factor. In other words in our formula the coefficient of F, functional ability for ocular function, must be

(1) and not a fraction for full vision and it must be 0 and not a fraction for total loss of vision. Thus it is demonstrated that the relationship of visual function to total function is that of an indispensable element; and being indispensable its mathematical relation to the entire bodily function is that of an integral factor which is a unit. The formula for visual earning ability then does not differ from that for full earning ability, but remains as in the original formula $F \times C = E$, and the primary coefficient for visual function is one (1)—one hundred per cent.

IV. The Measurement of Visual Function, Factor F.

We have shown above that in an economic sense the numerical value for the full function of vision is identical with that for total functional ability. Therefore this fact becomes our basis for evaluation of the visual function—that is 100%. Now in order to determine the percentage loss for any partial loss of vision, we must proceed by analysis as we did in our consideration of the earning ability, to determine in a logical way what elements or factors enter into the normal visual act, which elements are essential, and what is the relation of each essential element to the other. By such an analysis we find that vision is a complex act, that some factors or elements in this act are indispensable to normal vision and that others are not indispensable although important. There is a general agreement that the essential or indispensable elements of vision are (1) perception (visual acuity), (2) allocation (field function), and (3) muscular movements. That these elements are essential is fully discussed in *Transactions of Section on Ophthalmology, A.M.A.*, 1922, pp. 11–12. Among the non-essential functions we find color perception, depth perception, orientation, adaptation to light and dark, fusion sense, etc.

A consideration of each of the three indispensable elements of vision reveals that in an economic sense not only is each of these elements essential to normal visual function, but also that the action of each is co-ordinate and reciprocal. However, it cannot be said that each of these three factors is of equal importance, for we all know that visual perception

(or visual acuity) is by far the most important. How, then may we compute the numerical value of these separate elements which compose the complete act of vision, which shall satisfy these conditions? Again we find that by the law of the relation of indispensable co-ordinating elements of complex organisms, these elements stand in the relationship to one another as do the factors in a product and, undamaged, they are each of unit value. If any element is more important than another, such an element may be given a root value since by so doing its partial loss has less influence on the total product, which is the thing desired; but this mathematical treatment does not alter the relation of these elements as factors of a product nor does it conflict with our premise that the product of the combined essential factors is one (1) or 100%, because the square root of one is one. Neither does it conflict with the premise that the product is 0 when any element is lost, since the square root of 0 is 0. Therefore field function may be given a root value since it is less important than acuity.

For the purpose of brevity and clarity, let P represent perception (acuity of vision), A allocation (field), and M muscle function, and let F represent full vision; (F now representing visual function being synonymous with total bodily function). Then the formula for full vision would be,

$$P \times \sqrt{A} \times M = F.$$

And, since the coefficient of each element for full normal vision must be one in order to obtain a 100% product, and since the $\sqrt{1}$ is 1, then our numerical formula for complete normal vision is

$$1P \times \sqrt{1A} \times 1M = 1F \text{ or } 100\% \text{ vision.}$$

When there has been total loss to one or more factors, the formula for expressing the amount of vision would be for total loss of any one factor as follows:

For perception, $F = 0 \times 1 \times 1 = 0$
For allocation, $F = 1 \times 0 \times 1 = 0$
For muscle function, $F = 1 \times 1 \times 0 = 0$

The loss of two elements would of course give a 0 product. These formulæ, then, completely satisfy our premise in this

respect—that the total loss of any indispensable factor causes a total loss of function.

Having established the numerical value of each factor and their relationship to one another for no loss and for total loss of one or more, the next step in our problem is to determine the percentage of functional loss when there occurs a partial loss of one or more of these essential functions of vision. It must be obvious that when there is a partial loss to visual acuity, or to field, or to muscle function, the coefficient of any of these factors must be a fraction or some percentage of the whole, depending on the degree of disturbance of any of these elements. In order to determine the coefficient for each we must measure the disturbance of each by accepted standards which will give us the percentage of loss for each factor. We shall reverse the order in the discussions of this phase of our subject, leaving the most important factor, that of visual perception, to be discussed last and presenting our discussion of the measurement of muscle function first.

Coefficient of Factor M.—The value of the muscle function may be determined in a practical way. In the first place it must be admitted that any paralysis which causes a constant irremediable diplopia causes the complete loss of use of all the functions of one eye. It is so regarded by the Compensation Law of New York State. From a practical point of view most of the cases of muscle disturbance due to industrial injury or disease are either curable or incurable, the latter usually causing irremediable diplopia and the loss of binocular single vision. When this occurs it should be regarded, as it is in New York State, as having the same value as that for the loss of one eye. It is also regarded so by Magnus. When an irremediable diplopia necessitates that one eye be closed constantly, either voluntarily or by the use of a covering, it can have no economic value. Or should one learn to suppress the double image there would still be the loss of useful function. Therefore, the measure for the loss of muscle function which causes irremediable diplopia is equal to the loss of use of one eye and the coefficient for this factor is o when one eye only is being considered. In other cases the amount of functional loss and the coefficient for partial diplopia may be estimated in proportion to the area of the motor field in which diplopia is persistent

considering also the usefulness of that part of the motor field. Diplopia in the lower half of the motor field is just as serious as is a complete diplopia, whereas diplopia in the upper motor field disturbs visual working ability only slightly. In general a permanent paralysis of any single extrinsic ocular muscle causes diplopia in one half of the motor field. Thus in these cases the coefficient of factor M would be .50 (.50 M).

Coefficient of Factor A.—The numerical independent value for partial loss of field vision is determined by the amount or degree of concentric contraction. This concentric contraction may be anywhere between the 65 degree meridian (no loss) and the 5 degree meridan (total loss of field). The co-ordinate factor value of field is the square root of this fraction which is the coefficient of factor A, allocation. The percentage of field function remaining after various degrees of contraction and the square root of this quantity is shown below:

TABLE I. SHOWING THE COEFFICIENT VALUE OF FACTOR A. (FIELD).

Contraction to		*Per Cent. of Normal Field*		*Coefficient of A*
65°	=	100,	square root of =	1.00
60°	=	92,	" " " =	.96
55°	=	83,	" " " =	.91
50°	=	72,	" " " =	.86
45°	=	67,	" " " =	.81
40°	=	59,	" " " =	.76
35°	=	50,	" " " =	.71
30°	=	42,	" " " =	.65
25°	=	33,	" " " =	.58
20°	=	25,	" " " =	.50
15°	=	16,	" " " =	.40
10°	=	8,	" " " =	.28
5°	=	0,	" " " =	.00

This method of assigning a root value to the field function does not violate the premises previously laid down, to wit, that no loss to field produces no loss to total function and that a total loss to it produces a total loss to visual function, because as previously stated the square root of 1 is 1 and the square root of 0 is 0. We find further that by giving to the field a root value there will be for the slight disturbance of field only

a slight loss to total visual function, and for the more serious losses there will be a greater proportionate loss to function, which is in exact harmony with what actually happens when there is a partial loss to field. As a matter of practical application the industrial cases in which there are slight disturbances of field are not very frequently met with. Most of the cases due to industrial accident are the hemianopsias. For the purpose of illustration let us consider a practical example of this condition.—An homonymous hemianopsia without disturbance of central acuity is a loss of one half of the field in each eye. By using the square root of .50 which is .71, we determine that there is 29 per cent. loss of visual function for each eye or 58 per cent. loss for both eyes. This is an equitable adjustment and is an approximate average of the amount estimated by several authorities. Compare Holt's[1] estimation for the same defect which is 54 to 62 per cent., or that of Magnus[2] which is 68 per cent.

Coefficient of Factor P.—The consideration of a mathematically correct method for the measurement of visual perception and partial loss thereto should have our careful attention since there is still a divergence of opinion in regard to the correct mathematical interpretation of the accepted Snellen standard of measurement for the different degrees of visual acuity. This is the most important phase of our problem. In the United States the Snellen 5 min. angle or $\frac{20}{20}$ letter has been accepted as the standard of normal visual acuity and we find that there is an almost universal agreement on accepting $\frac{200}{1200}$ as a standard for industrial blindness. Taking these extremes as standards, how may we correctly determine in a mathematical way the utility value of the various degrees of visual perception or acuity? Let us first consider some of the many suggestions which have been advanced by students of this problem.

Attention should be called to the fact that the Snellen expressions are merely characters which express in a scientific way certain observed facts. They are not numerical fractions which can be reduced, and no student of visual economics so

[1] *Am. Encyc. of Ophth.*, vol. xviii., p. 13633.
[2] *Visual Economics*, p. 121.

regards them. Herman Snellen[1] in 1897 pointed out the errors which arise when the Snellen equation is reduced to fractions, stating that this procedure "tends to produce a wrong impression of the proportionate values of the visual acuity which are indicated by the test." Green[2] in 1905 warned against the "erroneous interpretation" which occurred when the Snellen formula was reduced to a fractional form. Allport[3] has also pointed out that "erroneous and misleading" conclusions are arrived at when the "international language" of any Snellen expression is regarded as a common fraction.[4]

Visual acuity being measured by an inverse ratio, the whole amount of vision must be known before one can mathematically determine the percentage of any fractional part. For example, in determining the percentage value of $\frac{2}{4}\frac{0}{0}$ we must determine what fractional part of acuity $\frac{2}{4}\frac{0}{0}$ is of the whole. This is true also of any other Snellen expression. The measurement of visual acuity depends on the relative size of the visual angle.

There are now prevalent several methods of interpreting the value of the Snellen equation. One method is that of assuming that between the standards of $\frac{2}{2}\frac{0}{0}$, (5 min. angle) and $\frac{2}{2}\frac{0}{7}\frac{0}{5}$, (55 min. angle) vision falls or increases in arithmetical progression. Based on this assumption, since with each additional distance of 10 feet for Snellen character there is 2.5 min. increase in the size of the visual angle, which is an increase of 5% in the size of the visual angle, and, since vision falls in a decreasing ratio, there is a decrease of visual acuity of 5% for each 10 foot increase in the size of the Snellen letters and 10% for each increase of 20 feet Snellen (5 min. angle), 5 min. angle being $\frac{1}{10}$ of 50. (Between a 5 min. angle and a 55 min. angle there is just 50 min.). Thus $\frac{2}{4}\frac{0}{0}$ being a 10 min. angle is just $\frac{1}{10}$ or 10% of the whole, etc. This is the method proposed by the committee of the A.M.A.[5] for measuring visual acuity and

[1] *System of Diseases of the Eye*, Morris and Oliver, vol. ii., p. 15.

[2] *Trans. Am. Ophth. Society*, vol. x., p. 650.

[3] *J. A. M. A.*, vol. lxxiv., p. 167.

[4] In the Snellen expression the character above the line indicates the distance at which the test is made, and the character below the line indicates the distance at which that letter or character subtends a 5 min. angle.

[5] "Report of Com. on Eye Injuries, A. M. A.," *Trans. Sec. on Ophth.*, 1922, pages 339 and 344.

for determining the percentage for the various degrees of acuity from $\frac{1}{10}$ to $\frac{200}{200}$. Based on this same principle there are several other suggested systems which are founded on a different assumption for industrial blindness and different conceptions of the economic value of the test characters.

Dr. Holt considers it sufficiently accurate to regard the degree of loss to visual function as "slight ($\frac{20}{25}$ to $\frac{20}{35}$), severe ($\frac{20}{40}$ to $\frac{20}{60}$), nearly total ($\frac{20}{70}$ to $\frac{200}{180}$), and total $\frac{200}{200}$ to 0)," these standards being purely empirical, and he then gives a coefficient value to each of these of .03, .09, and .18, assuming that .18 is the coefficient value for total loss of function of one eye.

Magnus reduces the Snellen standards which he calls the "Scientific Standard" to an "Economic Standard" and makes two tables, the first being applied to those vocations which demand higher degrees of visual acuity, and the second being applied to those vocations which demand lesser degrees of visual acuity. The former gives a range of $\frac{1}{12}$ to $\frac{11}{12}$ depending on the visual limits of .2 to .7 ("scientific standard"), the latter from $\frac{1}{8}$ to $\frac{8}{8}$ depending on the visual limits of .1 to .45 ("scientific standard").

In the opinion of the writer the proportionate value of the different degrees of visual acuity is best and most accurately determined when it is based on the accepted and approved Snellen letters or charters, constructed and used in geometrical progression, regarding each step in the gradation as having equal value. The reasons for this conviction are:

1st.—Test letters constructed in geometrical progression have been in practical use by ophthalmologists throughout the world for more than fifty years, optotypes so constructed having received the stamp of approval of many scientific societies.

2d.—The proportionate value of acuity of vision based on tests made with test-letters constructed in geometrical progression preserves throughout the series the fundamental principle laid down by Snellen, that the *value* of the acuity of vision is the reciprocal of the smallest visual angle formed by the test-object that can be distinctly seen, this visual angle being measured by *comparing* the distance at which the letters are just seen with the distance at which they subtend an angle of fine minutes, the comparison being carried out in complete

sequence throughout the entire range of vision. In the geometrical series the interval between any two series of test-letters is constant so that acuity is always measured by the same ratio of increase, whether it be between the first and second test-letters or between the tenth and eleventh or any other two.

In regard to the first point we may call attention to the fact that there are in existence no series of test-types which contain the complete series of test-letters in arithmetical progression from the standard of $\frac{10}{10}$ to practical blindness of any standard. And if constructed any such series would be impractical and lead to false valuations. See those suggested by Higgins[1] as placed in graph III. Standard test-types in geometrical progression were first suggested in this country by Green.[2] These differed only slightly from those proposed by Snellen. Later, test-types in geometrical progression were accepted by the American Ophthalmological Society[3] as "standard." Likewise letters in geometrical proportion were approved by the Fourth International Ophthalmological Congress, London, 1872, and they were also accepted by the *Société française d'Ophtalmologie.*[4] Test-objects in geometrical progression have also been indorsed by such well-known scientists as Herman Snellen[5] and Landolt[6] as well as many others.

In regard to the second point I find, as stated by Green, "It is the property of any series of optotypes in geometrical progression that the ratio of the visual angle subtended by any letter to that subtended by any smaller or larger letter is constant for all distances." The common ratio is 1.26. For method of construction of test-letters in geometrical progression the reader is referred to the *Transactions of the Am. Ophth. Soc.*, vol. x., p. 649–654, from which the above

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[1] Higgins, S. J. Report to the Wis. Industrial Commission, read before the Chicago Ophth. Society, May, 1919.

[2] *Trans. Am. Ophth. Society*, 1867, p. 68.

[3] Green: Standard Test-types, *Trans. Am. Ophth. Society*, vol. x., p. 190, also *idem*, vol. x., p. 644.

[4] *Annals d'Oculistique*, Mai, 1904, p. 400.

[5] Norris & Oliver, *System of Diseases of the Eye*, vol. iv., p. 16.

[6] *Refraction and Accommodation*, Landolt, p. 230.

quotation is abstracted. A careful consideration from a practical point of view of the manner in which visual acuity falls between the standard of perfect vision, $\frac{20}{20}$, a 5 min. angle, and total blindness, fingers at one foot or any other standard (the standard for blindness of the Missouri State Blind Pension Law, 1921, is $\frac{20}{400}$, a 115 min. angle) will clearly show that acuity of vision does not fall by equal gradations proportionate with the arithmetical ratio of increase in size of visual angle. Assuming that acuity does fall in an arithmetical ratio, and taking as the standard for blindness, that of the Missouri Statute for Blind Pension, $\frac{20}{400}$, we would find that this would make $\frac{20}{220}$ better than fifty per cent. of visual acuity, since $\frac{20}{220}$ is less than one half the size of the $\frac{20}{400}$ letter. The error of this assumption is obvious when we consider that it is the consensus of opinion that $\frac{20}{220}$ should be regarded as "industrial blindness." Thus it may be seen that visual acuity does not fall proportionately with each increase of 2.5 min. of the visual angle from normal vision to blindness. Or to state the same proposition in more familiar terms, the increase of 10 feet from $\frac{20}{20}$, $\frac{20}{30}$, $\frac{20}{40}$ in complete series through to $\frac{20}{320}$, $\frac{20}{380}$, $\frac{20}{340}$, etc., does not express an equal proportionate decrease in visual acuity. For example, it is indisputable that a decrease in visual acuity from $\frac{20}{20}$ to $\frac{20}{40}$ is much greater than is a decrease from $\frac{20}{320}$ to $\frac{20}{340}$. This is known from practical experience. Also one may see that, although the size of the angle is 5 min. larger in each instance, in the first instance the $\frac{20}{40}$ letters (10 min. angle) are twice as large or 100 per cent. larger than the $\frac{20}{20}$ letters (5 min. angle), whereas in the second instance the $\frac{20}{340}$ (85 min. angle) letter, is only .062 or 6 per cent. larger than the $\frac{20}{320}$ (80 min. angle) letter. Vision measured by a 6 per cent. difference between two angles can not be equal to that measured by a 100 per cent. difference between two angles. In the geometrical ratio vision is always proportionate to the relative size of the visual angle and all ratios are equal, $\frac{20}{40}:\frac{20}{60} = \frac{20}{120}:\frac{20}{240}$, etc. For example, also see graph II, the $\frac{20}{40}$ letter is twice as large as the $\frac{20}{80}$, and the $\frac{20}{120}$ letter is twice as large as the $\frac{20}{60}$, and the $\frac{20}{240}$ is twice as large as the $\frac{20}{120}$. Therefore the percentage of visual acuity between these points is the same. Note on the graph that between $\frac{20}{40}$ and $\frac{20}{120}$ acuity falls 30 per cent., and between $\frac{20}{120}$ and $\frac{20}{240}$ it falls 30 per cent.

That is, visual acuity falls in direct proportion to the relative size of the visual angle or size of the test character, which principle is consistent with our fundamental concept that vision falls in acuity proportionately with the size of the visible image. Constructing a graph on the principle of geometrical progression, considering each gradation of equal fractional value, and basing the percentage on the principle that visual acuity is in inverse proportion to the size of the retinal image, we find that the line which represents the decrease or fall in visual acuity is a symmetrical curve and not a straight line,

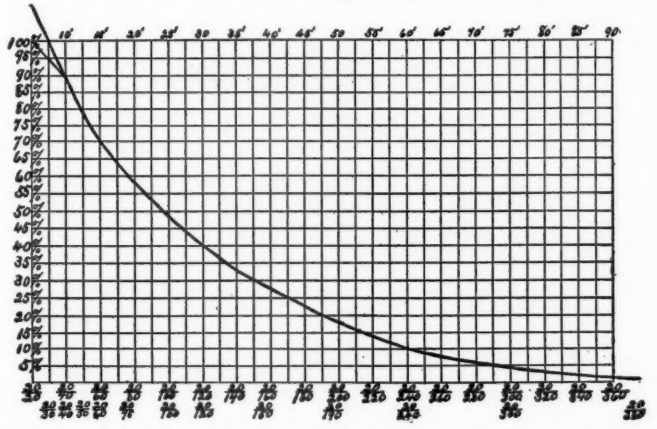

FIG. 2. Curve for visual acuity in uniform ratio, making equal gradations from $\frac{20}{10}$ to $\frac{20}{200}$ and corresponding percentages of visual acuity.

that such a curve falls uniformly, and that it represents vision, when it approaches the degree of blindness, as falling gradually to $\frac{20}{400}$ or beyond, depicting a state of affairs as they actually exist. Any conception or graph which considers or represents visual acuity as falling in a straight line and becoming nil suddenly, for example at $\frac{20}{220}$, or at $\frac{20}{800}$, or at $\frac{20}{400}$, etc., does not represent the true behavior of falling vision. See graph III.

The mathematical values for the different degrees of visual acuity computed in accordance with the above principles are shown in the following table. These may also be found by consulting the graph, the ordinates indicating the Snellen

letters and also the corresponding visual angle, while the abscisses indicate the percentage of visual acuity. These percentages are the coefficients for factor P in our formula for determining the utility of the complete act of vision. .

TABLE II.—SHOWING THE CORRESPONDING FRACTIONAL VALUE OF THE SNELLEN EXPRESSION, BASED ON EQUAL GRADATIONS FROM $\frac{20}{10}$ TO $\frac{20}{800}$.

Snellen Expression	*Fractional Value*	*Percentage and Coefficient for P.*
$\frac{20}{20}$	$\frac{10}{10}$	100
$\frac{20}{30}$	$\frac{9.5}{10}$	95
$\frac{20}{40}$	$\frac{9}{10}$	90
$\frac{20}{50}$	$\frac{8}{10}$	80
$\frac{20}{60}$	$\frac{7}{10}$	70
$\frac{20}{75}$	$\frac{6}{10}$	60
$\frac{20}{100}$	$\frac{5}{10}$	50
$\frac{20}{120}$	$\frac{4}{10}$	40
$\frac{20}{160}$	$\frac{3}{10}$	30
$\frac{20}{200}$	$\frac{2}{10}$	20
$\frac{20}{240}$	$\frac{1}{10}$	10
$\frac{20}{800}$	$\frac{0}{10}$	0

Thus, when there is a partial loss to one or more of the essential factors of vision,—muscle function, field, or visual acuity,—the coefficient for each may be determined separately and the total value of full visual function is the product obtained by multiplying all of these coefficients together, and this product in turn becomes the coefficient of factor F in the formula representing earning ability:

$$(P \times A \times M)\ F \times C = E.$$

For example, in order to determine full visual function and the coefficient of F when visual acuity alone is less than normal, assuming $\frac{20}{80}$ Snellen, no disturbance of muscle or of field, we find that $\frac{20}{80}$ Snellen is 80 per cent. of standard visual acuity, so .80 is the coefficient of P, and the coefficients of A and of M are 1, these factors being normal. Therefore, by multiplying together these coefficients we have the formula for full

vision, .80 × 1 × 1 = .80 or 80 per cent. visual function, and this becomes the coefficient of F. If at the same time that visual acuity is $\frac{2}{20}$, the field were contracted to 35 degrees, by consulting the table we find that for this degree of contraction, the coefficient of factor A is .71, then for full vision we have the formula, .80 × .71 × 1 = .568 or 57 per cent. visual function this being the coefficient for factor F. And should there be in addition to these disturbances a paralysis of one ocular muscle, the coefficient of M would be .50 and our formula would be

.80P × .71A × .50M = .284, or 28 per cent. vision.

(To be continued.)

FACIAL HERPES CORRELATED WITH FAULTY OCULO-MUSCULAR ADJUSTMENT.[1]

By Dr. CHARLES W. STEVENS, New York.

(With one illustration on Text-Plate VI.)

The case which I wish to report is one of a skin eruption of the forehead and temples, herpetic in character, and correlated with faulty oculo-muscular adjustment.

The patient was a young woman twenty-three years of age who first consulted me in April, 1920. She stated that since childhood she had been subject to severe headaches accompanied with vertigo and nausea. They were sudden in their onset, frequent in occurrence and lasted from a half to an entire day. She also stated that when she was about nine years of age she had had chorea and that even now she had occasional twitchings of her face and hands.

It was also noted that there was an eruption of small vesicles on the temples and forehead extending upwards just beyond the hair line. These were arranged in groups and would appear whenever she had a headache and recede shortly after recovery. They appeared sometimes on one side, sometimes on the other, and again on both sides at once. They were accompanied with pain and severe itching. The headaches and accompanying skin disturbance occurred at least once a week and sometimes oftener.

A functional examination of the eyes revealed the following conditions: Vision right eye $\frac{6}{6}$ with Cyl. + 3.00 D, Axis 115°; left eye $\frac{6}{6}$ with Cyl. + 3.00 D, Axis 65°. There was no heterophoria. The rotations of the eyes were 32° upward,

[1] Presented before Section on Ophthalmology, New York Academy of Medicine, May, 1923.

ILLUSTRATING DR. CHARLES W. STEVENS' ARTICLE ON "FACIAL HERPES CORRELATED WITH FAULTY OCULO-MUSCULAR ADJUSTMENT"

40° downward, 48° inward, and 38° outward. The clinoscope showed declination right +4°, left +2°. On subsequent examinations this declination varied slightly showing sometimes more on the left side than on the right, thus indicating that there was about an equal amount of positive declination in both eyes.

The refractive error had already been properly corrected before she came to me. As there was no heterophoria and as the rotations, although slightly restricted in all directions, were proportionately normal, it was evident that the case was a simple one of faulty relationship of the vertical meridians of the retinæ, that is declination.

A series of surgical operations, extendo-contractions, was undertaken with the purpose of correcting the faulty leaning of the eyes. As the work progressed there was a gradual improvement in the headaches; they came at less frequent intervals and were less severe in character. There was also noted an improvement in the extent and character of the eruption on the face.

The patient called at my office in April of the present year (1923) after an absence of about sixteen months. She reported that she had been practically free of headaches for over a year and that the herpetic eruption of the face had disappeared. An examination at that time showed again no heterophoria, the rotations as at the first examination and the declination reduced to + 1° in each eye, an amount which as a rule will cause no special disturbance.

Several interesting conclusions may be drawn from this case.

It will be noted that of the three fundamental adjustments of the eyes, the relationship of the lines of regard, the relationship of the plane of regard, and the relationship of the corresponding retinal meridians, it was the last only which was unfavorable, and it was through the correction of this adjustment alone that relief from the nervous symptoms was accomplished. This fact should emphasize the necessity in every functional examination of the eyes of carefully investigating all of the ocular adjustments which are involved in the proper maintenance of easy binocular vision. A failure in this case to have made an examination with the clinoscope would

have meant a failure to comprehend the true source of the nervous irritation.

An examination of a photograph taken when the patient first came to me reveals certain significant facial characteristics. We notice in particular the highly arched eyebrows with the integument drawn up and back and tense over the supraorbital arches and the superciliary region. This particular form of facial expression is typical of that very frequently associated with a double positive declination. The mechanism of this is very simple. The temporal leaning of the vertical meridians is best controlled by the superior obliques. But these muscles by their overaction would cause a divergence of the eyes, and in many cases they do, thus producing an exophoria. Hence the internal recti are called upon to prevent this divergence and in order to free the inner canthus of any possible impediment to easy action of these muscles the eyebrows are raised and the whole musculature of the forehead is drawn upward. Of course the lower facial muscles assist in freeing the inner canthus but in the present case we are not particularly interested in their action.

Now it is hardly possible to think of this constant tension of the musculature of the forehead without realizing that it must have some effect on the terminal branches of the frontal nerve which ramify through all the tissues of this region. Pressure will undoubtedly be exerted upon them owing to the thinness of the tissues and the unyielding bony structures beneath. This pressure may occur either at the point where the nerves emerge from the orbit over the supraorbital arches or at any point in their further course.

Dr. Robert W. MacKenna in his recent work *Diseases of the Skin* makes this statement: "In general it may be said that any condition capable of provoking neuritis may be a proximal cause of an attack of herpes." Continued pressure can certainly be considered as a cause for neuritis and hence as a cause of herpes. I am also informed by a distinguished dermatologist that abdominal herpes is often seen as the result of wearing a belt that is too tight. Therefore if pressure on the abdomen will result in herpes there is every reason to assume that it will have the same effect on the forehead where anatomical conditions are far more

favorable to manifestations of nervous irritation from this cause.

We thus have a logical explanation of the correlation of the facial herpes with the faulty eye adjustment. As original cause there is the unfavorable relationship of the retinal meridians, the declination. The musculature of the forehead, called upon to assist in counteracting conditions which would be unfavorable to binocular vision, is kept in a state of constant tension. As a result of the tension, pressure on the nervous tissue causes irritation which in this particular instance was manifested by the vesicular eruption, or herpes simplex.

The most conclusive evidence of this correlation rests in the fact that correction of the unfavorable eye conditions has resulted not only in relief from the headaches but also in a disappearance of the skin eruption without any special dermatological treatment.

REPORT OF THE TRANSACTIONS OF THE OPHTHALMOLOGICAL SECTION OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. H. DICKINSON, London.

A meeting of the Section (the first in the new Session) was held on October 12th, the President of the Section, Mr. A. L. Whitehead, of Leeds, occupying the chair.

The Late Mr. Gustavus Hartridge.

The President expressed the sense of loss felt by members of the specialty at the death of Mr. Gustavus Hartridge, whose treatise on eye disease was so well known that it was practically a household word wherever ophthalmology was practiced. Deep sympathy with his surviving relatives was evidenced in the remarks.

Cases Shown.

Cysts of Conjunctiva.

Mr. J. F. Cunningham showed a patient with cysts of the conjunctiva. Apparently the condition began as an episcleritis 2½ to 3 years ago, and later the swellings now seen occurred. Col. Herbert, who saw the patient as he was a Ministry of Pensions case, had an examination of the general condition made, and the Wassermann and tubercular complement tests were negative. Col. Herbert had excised and examined a portion of a cyst, and reported it to be full of lymphocytes; he thought it an early stage of hyaline or colloid degeneration following upon episcleritis. The exhibitor asked for opinions as to treatment by radium or X-rays.

Mr. N. B. HARMAN suggested trying drainage by means of silk, as carried out by Mr. Sampson Handley in cases of cancer of the breast. In a case of fractured jaw followed by orbital œdema under the speaker's care this measure reduced the swelling a great deal.

THE PRESIDENT thought there might be some block at the back of the orbits, and a skiagram would be useful to determine that.

Mr. MALCOLM HEPBURN considered that if the President's suggestion were correct the œdema would by now have been more generalized. He thought it might be a brawny episcleritis. A similar—but not so extensive—case of his own was greatly benefited by diamine, after a number of things had failed.

Tumor of the Limbus.

Mr. F. JULER exhibited a woman aged 66 who had a tumor of the limbus, which had been present nine years. Three and one half years ago her tumor was taken away at a hospital, and since then a recurrence had gradually come on. In the portion of growth and from the pre-auricular gland the cells were of carcinomatous type.

Mr. E. TREACHER COLLINS spoke of the rarity with which primary epibulbar growths showed glandular involvement. In the present case he advised excision of the eye; it was not safe to trust to mere removal of the growth and the subsequent application of radium. In a less advanced case of his own he took the tumor away and then applied radium, and the patient survived several years thereafter, and eventually died of a different malady.

THE PRESIDENT spoke of three cases of the kind he had had under treatment. In two he exenterated the orbit, in the other he carried out the treatment just referred to by Mr. Collins. In the latter case the patient remained free from recurrence a number of years, and then died of chronic bronchitis. In none of those three cases was there any involvement of glands. He agreed with Mr. Collins' advice in the present case.

Mr. T. HARRISON BUTLER referred to a case of epibulbar carcinoma on which he operated fifteen years ago. That

patient remained well eleven years, then there was a recurrence. The patient did not come back for some time after that, but a removal of the recurrence was carried out. For a still later recurrence he handed the patient over to a general surgeon, and the ultimate result he did not know.

Epibulbar Melanotic Sarcoma.

Mr. WHITING showed a patient with this condition. There were also two pigmented areas on the lids, and the question arose whether there was a connection between these areas and the prime growth. Probably in the whole conjunctival tract there was a tendency to abnormal pigmentation. Clearly in this case the whole of the conjunctival tract should be removed, and the lid margins dealt with.

Arterio-Venous Aneurysm in a Boy.

Mr. N. BISHOP HARMAN showed a boy, aged 12, who, two years ago, was looking through railings when another boy poked some wire into his eye. It entered the inner canthus, but had left no marks. In July the eye became markedly proptosed, and there was a thrill and a bruit. The conjunctiva was not injected, however, and the fundus was normal and the vision $\frac{6}{9}$. The continual hissing noise was said to have prevented the boy from sleeping. In a quiet ward it could be heard by the observer without the aid of a stethoscope. With a stethoscope the bruit could be traced down below the angle of the scapula. The muscle balance was good; there was a trifle of hyperphoria, and one degree of esophoria. The media were normal, but the left physiological cup was deeper than the right. Pressure on the eye with an elastic pad made it recede into the orbit, but not to the same extent as the right eye. The physician reported that there was no valve disease, and no diastolic murmur in any posture. The blood pressure was within the normal range. He had suggested that the surgeon should tie the internal or the common carotid.

Mr. BASIL LANG spoke of the condition when he saw the boy

at an earlier stage of his condition. There was then proptosis, but very little bruit.

Mr. TREACHER COLLINS said Mr. Power advocated ophthalmic surgeons being familiar with the operation of tying the carotids, as sometimes it was required as a matter of urgency. In one traumatic case of his own, in which a bullet was beneath the basilar process, the common carotid was ligatured by a friend. There was immense relief for a year, and then the noise began to return.

THE PRESIDENT said he had seen four of such cases, and in each he had himself tied the common carotid. One was a case of trauma, following a fall, another was due to gunshot injury when rabbit-shooting, and the other two were syphilitic, and an ordinary syphilitic aneurysm had apparently burst through into the cavernous sinus. In three of the four cases the measure he carried out was satisfactory and produced permanent relief. In one of the cases some of the proptosis returned, but not the bruit. In all the cases the vision had been considerably impaired before the operation, and this did not improve afterwards. Mr. Harman's case still had $\frac{6}{9}$ vision, and ligature of the common carotid was certainly indicated.

Mr. R. FOSTER MOORE reminded the Section of the discussion on this subject at the last Ophthalmological Society's Congress, last May.

Sir JOHN PARSONS referred to the collection of records of these cases made by De Schweinitz and Holloway, showing that all the methods of ligature carried out could claim some successes. The great thing to avoid was tying either both externals or both commons at the same time, for cerebral anæmia was then likely to quickly supervene; sufficient time must be allowed between the two operations to allow of some collateral circulation to be set up. There was a consensus of opinion that it was better to tie the internal or the external carotid than the common, though it was more difficult to do so. In some of the cases in which there was a recurrence and big veins in the lids and in the neighborhood of the eye, the veins were ligatured, then excised. In males the cases were mostly traumatic, while practically all the cases in females were syphilitic.

A New Method of Photographing a Foreign Body in the Anterior Part of the Eye.

Mr. T. HARRISON BUTLER exhibited a skiagram of a foreign body in the anterior part of the eye, which was taken by an improved method. It was notoriously difficult to photograph these foreign bodies in the ordinary way through the skull. The idea originated with the late Sir James Mackenzie Davidson, and had been elaborated by Professor Foch, of Zurich. A small dental plate is pressed as far as the patient would permit between the eye and the orbit; the X-ray tube was placed on one side, and an exposure of three seconds given. Then a further exposure was taken from below. A small fishhook on the conjunctiva provided the localization. The foreign body in this case was probably lead; the magnet had been unsuccessfully tried, and the accident followed hammering on a lead-faced vice in an engineer's shop.

Mr. BERNARD CRIDLAND spoke of the value of the method in one case in which he had recently tried it.

Muscle Recession in Strabismus.

Mr. T. HARRISON BUTLER read a paper on this subject, intended as an introduction to a discussion. He said the most important object in a squint operation was a cosmetic one, to get the eyes parallel; the second was to help in the cure of the amblyopia. There was an opinion prevalent—and it was largely true—that the amblyopia came on only in youth, and that this only was the time of life when it could be cured. But he had seen a number of cases in which definite amblyopia developed when the child was over ten years of age. Most of the squint cases on which he had operated had from 30 to 45 deg. of squint. Eight he regarded as the best age for the operation; the child was less trouble at this age than younger, and the condition was not too firmly established. In squints of 45 deg. which had been in existence a number of years, he suggested there was not only disturbance of the fusion faculty, but in some cases there was definite anatomical alteration. In some cases the internal rectus was thickened, and showed signs of contracture. One could not hope to cure a squint

of over 30 deg. by operating on any one muscle, and one was thrown back on something like tenotomy. But some of the cases diverged widely at a later stage, and a second advancement was rendered necessary. Therefore he had done recession of the internal rectus. He had simplified Jameson's operation, described in the ARCHIVES OF OPHTHALMOLOGY. By means of a drawing he illustrated the successive steps of the operation as he now performed it. He used one suture, instead of Jameson's three. In conclusion, he discussed the Tucker operation.

Mr. CLAUDE WORTH said that if a child had squinted more than half its life, as a rule it could not be remedied by merely strapping up the fixing eye. Usually, the maximum age for the operation could be put at 7 or 8 years. In a large experience, it was very rare for him to see a case in which the internal rectus was either shortened or too strong, though he admitted there were rare cases of **strabismus fixus.** If an eye was quite amblyopic, it was advisable, if there was good balance, to operate on only one eye. He regarded as bad all tenotomies, some were worse than others. One reason he said was that he rarely met with a case in which he was satisfied the muscle was too strong, therefore why weaken it? Another reason was that if tenotomy had been done, it made accuracy in operating impossible, as one did not know what the effect of that tenotomy would be. Those who had done advancement with certainty would not revert to tenotomy.

Mr. N. BISHOP HARMAN spoke concerning tenotomies. The risk, in children especially, of putting a suture on the sclera, on the inside, was very great, as there was so little room. He did not agree there was any alteration in the internal rectus in cases in which the external rectus appeared to be thin. In Tucker's operation, the success largely lay in thoroughly rasping the surfaces of the tendon. He performed many operations for squint, receiving a good number of cases from school clinics, and he always did the reefing sub-conjunctivally. He objected to the use of catgut in these operations, as it must be absorbed, and hence there must be an invasion of leucocytes, and a granuloma formed. He had excised such because it gave an unsightly appearance.

Mr. GRIFFITH said he had done an operation like that de-

scribed by Mr. Harrison Butler for the last eighteen months. He gave up simple tenotomy because he had been dissatisfied with the uncertain results; there were some late divergences, and then he took to doing advancements. When the patient was under the influence of the cocaine, he seized the eye and abducted it, judging the degree to which the internal rectus was resisting. If it was easy, he did an advancement; if not, he did a retirement of the internal rectus. The latter operation was not as easy as an advancement. Having dealt with the muscle in long-standing squint, he did a plastic operation, sewing it up so that the vertical incision became a horizontal one.

Mr. BUTLER replied.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. BEN WITT KEY, Secretary.

MEETING OF MAY 21, 1923. DR. ELLICE M. ALGER, CHAIRMAN

Dr. J. M. Wheeler presented a patient showing **complete inversion of the iris.** February 15, 1923, the right eye was struck violently by the end of an iron rod about three feet long and one-quarter inch in diameter. The patient was first seen four days after the accident, suffering from extreme photophobia, blepharospasm, and lacrimation. There was a small flap of partially detached cornea at the site of a perforating wound. Removal of this flap and curetting of the corneal wound greatly relieved the discomfort. At the first examination, there was no blood in the anterior chamber and no iris could be seen. Vision was $\frac{20}{200}$ and the visual field good. Tension was normal and the eye quiet. A corneal scar marked the site of the wound. The lens was partially cataractous but not tremulous. There was a defect on the nasal side, where there appeared to be a wound of the capsule. The interior could be seen with difficulty and appeared normal. Operation was not advised.

Two cases of disease of the central nervous system complicated by the **syndrome of Horner** were reported by Dr. G. A. Blakeslee.

The first case was one of the syndrome of Avellis, complicated by the syndrome of Horner and dissociated sensory changes of the contralateral trigeminal area. This patient was a man, white, aged 56 years, single, and in good health until March 16, 1923, when he noticed a partial closing of the left

eye. On March 17, 1923, late in the day he began to have difficulty in swallowing, with a regurgitation of fluids through the nose. On the same day his voice changed to a whisper. On March 18, 1923, he noticed that the wind blowing through the window of his room felt warm on the right side of his face and cool on the left side. At this time when bathing he noticed that cold water felt warm on the right side of his face and the right upper and lower extremities.

Neurological examination.—Slight ptosis of left upper lid, myosis of left pupil, enophthalmos of the left eye, narrowing of the left palpebral fissure, and a paralysis of accommodation of the left eye. Light, convergence, and consensual reflexes were normal. This symptom complex is called the syndrome of Horner.

There was a paralysis of the left soft palate and left vocal cord of the lower motor neurone type and loss of pain and temperature sense of the right side of the body. The palatoplegia and laryngoplegia localized the lesion in the medulla oblongata at the level of the nucleus ambiguus which supplies the palate and larynx. The spinal fillet and secondary trigeminal tract lie close to the nucleus ambiguus and account for the pain and temperature changes on the contralateral side of the body. The syndrome of Horner is accounted for by the lesion involving the oculo-pupillary center of the medulla, which consists of a group of cells situated dorsal to the nucleus ambiguus at this level.

A few drops of a 4% solution of cocaine were instilled in each eye with the result that the right palpebral fissure widened, the pupil of the right eye widely dilated, and there was marked exophthalmos of the right eyeball. There was no change in the left eye, showing a cervical sympathetic paralysis. The lesion is probably due to a thrombus in one of the radicular branches of the left inferior cerebellar artery supplying the medulla oblongata.

The second case presented was one of syringomyelia and syringobulbia complicated by the syndrome of Horner. The patient was a woman, white, aged 34 years, married, and in good health until May, 1921, when she complained of hoarseness and prolonged coughing attacks. Several months later there was difficulty in swallowing and regurgita-

tion of fluids through the nose. May, 1922, she began to complain of weakness of the left arm and leg; also a sensation described as freezing, numb, or dead feeling in the left arm. About this time she received a severe burn on the left side of the neck from a nursing bottle, but was unaware of the burn until told of it later.

Neurological examination.—Left palpebral fissure was narrower than the right, slight enophthalmos of the left eyeball, myosis of the left pupil and a slight ptosis of the left upper eyelid. There was also paresis of accommodation of the left eye. Reaction to light and convergence, and the consensual reaction were normal. There was paralysis of the left soft palate and left vocal cord of the lower motor neurone type, with a loss of the palatal reflex on the left side. There was diminution of pain and temperature sense from the second to the eighth cervical segments on the left and right. This diminution was present in the left trigeminal area. The deep reflexes were lost in both upper extremities with slight atrophy of the small muscles of the left hand. Deep reflexes were increased in the left lower extremity. There was a Babinski on the left.

The paralysis of the palate and vocal cord on the left localized the lesion at the level of the nucleus ambiguus on the homolateral side. The root of the spinal Vth lies in close proximity to the nucleus ambiguus and may well account for the dissociated sensory changes of the trigeminal area. The bilateral dissociated sensory changes of the upper extremities are accounted for by a lesion of the ventral commissure of the cervical cord. An extension of this lesion anteriorly accounts for the loss of reflexes in the upper extremities, and the involvement of the pyramidal tract on the left side would account for the Babinski on the left side.

The syndrome of Horner could well be explained by the lesion in the bulb or the one in the cervical cord. As in the first case the bulbar lesion might involve the oculo-pupillary center of the medulla which lies just dorsal to the nucleus ambiguus and account for the syndrome. A lesion in the cervical cord from the second to the eighth cervical segments could well account for the syndrome of Horner by involving the pupil-dilating fibers in their course from the bulb, or the

cilio-spinal center in the lower cervical region may be involved as is frequently seen in syringomyelia of the cervical cord.

When a 4% solution of cocaine was instilled in each eye, the reaction was slight in the left eye, showing a paresis of the sympathetic of the left side.

DISCUSSION: Dr. C. W. STEVENS has seen both cases reported by Dr. Blakeslee. Neither of them showed as marked symptoms as at previous examinations. Paralysis of the dilator, the pseudo-ptosis on the affected side and the enophthalmus on the opposite side were difficult to demonstrate. These cases frequently go first to the neurologist. Benedict's Syndrome, Millard-Gubler's Syndrome, Foville's Syndrome, and Horner's Syndrome (cilio-spinal center affected) all occur as result of lesions in and about the same region.

Dr. I. GOLDSTEIN had seen a case of Horner's Syndrome in which the radial pulse on the left side was absent, when both arms were raised upright.

Dr. M. J. SCHOENBERG asked Dr. Blakeslee if the pupils reacted to adrenalin.

Dr. BLAKESLEE had examined the patient for a cervical rib but it was not present. He had not tried the radial pulse with the arms raised. Adrenalin had not been instilled, cocaine only had been employed.

Dr. C. W. STEVENS reported a case of **facial herpes** associated with **faulty ocular adjustment.** (Published in this issue.)

DISCUSSION: Dr. M. J. SCHOENBERG asked how many of these cases had Dr. Stevens seen? Dr. Stevens replied that he had seen similar cases but not like the one reported. Dr. Schoenberg inquired,—why do we not see more of these cases if this is a fairly common symptom?

Dr. H. H. TYSON asked how many operations are necessary in these cases. Dr. Stevens stated that five operations on the two eyes were necessary in this case. Of course it would vary with the case.

A paper entitled, **chronic gonorrheal prostatitis,** a possible **etiologic factor** in certain **inflammations** of the eye was presented by Dr. A. H. Thomasson. This paper is published in full in the Nov. ARCHIVES, 1923.

DISCUSSION: Dr. A. KNAPP said we were indebted to Dr. Thomasson for presenting to the Section these interesting cases and this method of demonstrating the presence of gonococci in the secretion of the prostate—and thus showing that the gonococci live in the prostate over such a long period of time—30 years.

Dr. HOMER SMITH referred to irido-cyclitis due probably to toxins from intestinal conditions. He mentioned especially those which may be due to the colon bacillus.

Dr. G. H. BELL pointed out that in searching for focal infections we are apt to overlook the genito-urinary tract. He referred to a case of irido-cyclitis relieved after the seminal vesicles and prostate gland had been stripped. Culture revealed gonococci. The vitreous cleared, iritis subsided and vision improved from hand movements two months before to $\frac{20}{40}$. In obscure disease of the eye, with a positive history, culture from the prostate should be made.

Dr. THOMASSON said that the most interesting point in these cases is that the gonococcus remains alive and virulent in the prostate for so long a period of time.

The modern conception of exophthalmic goiter was the subject of a paper by Dr. E. F. Du Bois. He stated that we doubtless have made no important advances in our theories of exophthalmic goiter since the classical work of Magnus-Levy more than twenty years ago. No one has yet proved that there is a dysthyroidism rather than a simple hyperthyroidism, although there are certain manifestations which point toward an abnormal quality of thyroid secretion.

Most recent investigators emphasize the importance of Magnus-Levy's discovery that the basal metabolism determination is the best single guide to the severity and progress of the disease. It is not an infallible guide and should never be considered apart from the usual clinical examination. The classical eye symptoms seem to be growing less important in the diagnosis. They may be found in other conditions and are often absent in hyperthyroidism, especially, in subjects over 35 years of age. The diagnosis should be made before the exophthalmos becomes marked. Tachycardia is the most important symptom. It is present in practically all true cases. In a suspected case the diagnosis of hyperthyroidism is hardly

permissable if the pulse rate becomes normal when the patient is resting quietly or sleeping.

Recent work by Kessel, Lieb and their associates has proved that exophthalmic goiter shows a tendency toward spontaneous cure if the patient is kept quiet. Means has demonstrated the curative value of X-rays in about two-thirds of the cases. Most surgeons prefer thyroidectomies as giving the surest and quickest results.

As yet no really satisfactory explanation of the cause of the exophthalmos has been offered.

Dr. J. M. WHEELER showed a case to illustrate the result of operation for the protection of the eye in **exophthalmic goiter.** The patient (J. S.), February 21, 1922, complained of an inflamed left eye. A diagnosis of ulcer of the cornea due to exophthalmic goiter was made. Scars were found on each cornea, resulting probably from previous ulceration.

April 29, 1922, both lobes of the thyroid were operated upon, and since the thyroidectomy the patient had gained in weight and improved genera lly.

December 14, 1922, Dr. Wheeler shortened the left palpebral fissure about 10*mm* by tarsorrhaphy. One week later advancement of the orbicularis of upper and lower lids of the left eye was performed. In this operation the orbicularis of the outer half of each lid was exposed near the lid margin. Strips of orbicularis were dissected up and advanced for attachment to the periosteum at the margin of the orbit. Thus the palpebral fissure is lengthened and narrowed. Operations of the same sort were performed on the right side on Jan. 5, and March 8, 1923. As a result, the cornea of each eye has been sufficiently well protected so that there has been no further corneal trouble, and the appearance of the patient has been considerably improved.

When it has been considered that numerous eyes have been lost from ulceration of the cornea in Graves' Disease, the importance of some effective local means of handling the endangering eye condition is evident. This is apart from the importance of proper handling of the general condition. The operations suggested are in the formative stage.

DISCUSSION: Dr. W. E. BENEDICT (Rochester, Minn.)

stated that of most importance in these cases is the lessening of the degree and the lessening of the incidence of the exophthalmos. Malignant types are not common. Those well nourished take care of the cornea very well. When poorly nourished nothing seems to help them. Exophthalmos appears to be due to inflammation of the orbit. Removal of part of the orbital tissue has been attempted. He referred to a case in which Dr. W. R. Parker (Detroit) had performed such an operation.

Dr. E. E. BLAAUW (Buffalo) said the case report was interesting and that the measures taken should relieve somewhat the orbital congestion and give relief.

Dr. C. W. CUTLER said these cases should be diagnosed and treated early. We cannot tell when a case will become malignant. These are fortunately rare. The ophthalmologist is usually called in when the cornea is in jeopardy and when any operative procedure is inadvisable because of the great tachycardia. Is the Kuhnt conjunctival plastic operation advisable in cases of ulcer of the cornea from exophthalmos?

Dr. A. KNAPP said that success in dealing with these cases depends upon the toxæmia. He asked Dr. Du Bois as to the character of the exophthalmos in cases where the rest cure is administered, also what if any effect from this form of treatment?

Dr. H. HYMANN referred to the locomotion pulse in these cases—and the effective treatment from thyroxogen.

Dr. O. DIEN questioned the wisdom of waiting in these cases till corneal involvement begins and threatens destruction of the eye. Early diagnosis and prompt measures were advised.

Dr. DU BOIS in closing plead for closer study and care of these cases. Treatment of the general condition is generally advocated. Crile advises operation on most cases. At the Massachusetts General Hospital the X-ray is used in a large majority of cases. At Mt. Sinai the rest cure is the popular method of treatment. It depends usually on the case and the conditions of life of the individual as to which kind of treatment seems most effective. Operation is usually advised before excessive exophthalmos comes on. After exophthalmos

has been present for a long time, it does not subside entirely with any kind of treatment.

Dr. WHEELER stated that he did not present the case and operative measure employed with any intention that it is final, but hopes it may be improved upon.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

Edited by Matthias Lanckton Foster, M. D., F. A. C. S., New Rochelle, N. Y.

(*Continued from November issue.*)

IV.—THE ORBITS AND ACCESSORY SINUSES.

53. Beck. **Empyema of the accessory sinuses and retrobulbar neuritis.** *Monatschr. f. Ohrenheilk. u. Laryng.-Rhinol.*, 1921, p. 43.

54. Birnbacher. **Phlegmon of the orbit caused by periostitis of an incisor.** *Zeitschr. f. Augenheilkunde*, xlvi., p. 167.

55. Franklin, W. S., and Horner, W. D. **Hernia through Tenon's capsule with extrusion of orbital fat.** A birth injury. *American Journal of Ophthalmology*, August, 1922.

56. Fuchs. **Small inflammatory foci behind the eye.** *Zeitschr. f. Augenheilkunde*, xlvi., p. 157.

57. Hirsch, Cæsar. **Air embolus in the central artery of the retina after washing out the antrum.** *Zentralbl. f. Ophthalm. u. Grenzgeb*, v., p. 457.

58. Pascheff. **Actinomycotic tumor of the orbit.** *Klin. Monatsbl. f. Augenheilkunde*, lxvii., p. 302.

59. Raffin. **Syphilis of the orbit.** *Ibid.*, lxvi., p. 747.

60. Stargardt. **Bilateral inflammatory pseudotumor of the orbit.** *Ibid.*, lxvii., p. 107.

61. Steiner. **Implantation of fat after enucleation and exenteration of the eye.** *Ibid.*, lxvii., p. 267.

62. Vogel. **Diagnosis of diseases of the accessory sinuses.** *Deutsche med. Wochenschrift*, 1921, p. 958.

63. Weiss. **Discussion of Hirsch's paper "Air embolus in the central artery of the retina after washing out the antrum."** *Klin. Monatsbl. f. Augenheilkunde*, lxvi., p. 920.

FRANKLIN and HORNER (55, **Hernia through Tenon's capsule with extrusion of orbital fat**) report a case in which orbital fat was present under the conjunctiva pushing out the lid in each eye as the result of traumatism in forceps delivery. An incision was made through the overlying conjunctiva and the mass excised. The stump retracted and the rent in the septum was closed with a catgut suture. The result was entirely satisfactory.

STEINER (61, **Implantation of fat after enucleations and exenteration**) states that the implant was cast off in three per cent. of the enucleations and in eight and a half per cent. of the exenterations. The failures were probably due to the presence of microörganisms left behind by the formerly inflamed eyes. After about five years the implants had shrunken to half their original size in fifty per cent. of the cases. This was restored by injections of paraffine. The results of the operation are so good that, in spite of the longer convalescence, especially in children, Steiner believes that it should be employed in nearly every case. It is contraindicated only by septic processes in the orbit or the eyeball.

BIRNBACHER'S (54, **Phlegmon of the orbit caused by periostitis of an incisor**) patient had pain in the right outer upper incisor together with exophthalmos, swelling of the lids, chemosis, infiltration of the orbital tissues and fever. The swelling involved the cheek. The tooth was extracted and pus flowed freely from the alveolus. An incision down to the bone showed that the margin of the orbit at the inner canthus was rough, and from here a probe could be passed along a track to the diseased tooth. A cavity in the alveolar process was filled with fœtid detritus. The mucous membrane of the antrum showed a polypoid proliferation. Drainage and irrigation cured the phlegmon in fourteen days. The eye was lost through perforation of the cornea.

FUCHS (56, **Small inflammatory foci behind the eye**) found microscopically small infiltrates along the blood vessels, nerves, muscles and posterior ciliary nerves outside eyes which had not been freshly inflamed, and so could not have been the origin of the fresh infiltrates. The cause was not clear. The patient had often complained of pain behind the eye for which no explanation could be found.

PASCHEFF'S (58, **Actinomycotic tumor of the orbit**) patient was 62 years old. Twenty-five years before he received a contusion on the brow. Thirteen years later abscesses appeared about the orbit. Twenty-four years after the contusion exophthalmos appeared. Examination showed that a fibrous tumor had destroyed the lacrimal gland, and that the tumor was in part soft and suppurating. Scattered about were foci of polynuclear leucocytes and sclerosing connective tissue. Bacteriologically a gram positive actinomyces closely related to the streptothrix farsini bovis was found. Pascheff has found no record of this agent having been hitherto found in man.

RAFFIN (59, **Syphilis of the orbit**) reports two cases, the second of which is worthy of note. The patient denied having had syphilis and his Wassermann was negative. His middle turbinate was resected; healing took a very bad course. Neosalvarsan was administered and had a prompt effect. A short time later he had dacryocystitis with periostitis and caries of the nasal bones. Wassermann was now ++, and a rapid healing was again obtained through neosalvarsan. This case tends to confirm the theory that dacryocystitis often indicates a specific disease of the periosteum of the nasal bone.

In November, 1919, the right eye, which had always been bad, of STARGARDT'S (60, **Bilateral inflammatory pseudotumor of the orbit**) patient, 68 years old, became distinctly protuberant. The eyeball was slightly injected, but otherwise normal, except that movement upward and to the left was bad. The left eye was normal. Examination of the internal organs and of the accessory sinuses was negative, as was the Wassermann test. The exophthalmos increased in spite of potassic iodide. The sphincter pupillæ became paralyzed, the lids swollen. Kroenlein was performed but no tumor found. The swelling increased. X-rays were without effect. A corneal ulcer appeared in March, 1920, and in April the orbit was exenterated. Microscopical examination showed only small foci of lymphocytes and, in the marginal parts, plasma cells; nothing indicative of tuberculosis or syphilis. Early in February, 1921, the left eye became affected, it protruded, but otherwise was normal. Again the general examination, the nasal examination and the Wassermann were negative. The exophthalmos

and swelling of the lids increased until the cornea became ulcerated; the ulcer improved after the lids had been sewed together. The writer believes the trouble to have been an infection, but does not know the agent.

BECK (53, **Empyema of the accessory sinuses and retrobulbar neuritis**) had quite a number of cases of sinusitis examined ophthalmologically without knowledge on the part of the examiner of the existing condition. The examination was to determine mainly whether the blind spot was enlarged, and whether scotomata were present. The diagnosis of empyema of the accessory sinuses was based on the clinical evidence and the roentgenograph. Among 91 cases of empyema, including 44 of the ethmoid and antrum, only one case of unquestionable retrobulbar neuritis was detected. In five others very slight eye symptoms were detected. The result of his investigation convinced Beck that no eye condition characteristic of retrobulbar neuritis can be said to be prominent in empyema of the accessory sinuses.

HIRSCH (57, **Air embolus in the central artery of the retina after washing out the antrum**) observed the following case. After washing out the antrum the right eye became blind, the pupil dilated with no reaction. Ophthalmoscopic picture, one minute after the accident, gray discoloration of the retina, white papilla, white, empty arteries, bloodless veins. After two or three minutes all symptoms and disturbances disappeared.

WEISS (63, **Discussion of Hirsch's case**) believes that the trouble was not an air embolus, but a vascular spasm set up through irritation of the vagus and sympathetic. In 1912 he saw a transient spasm in a person with arteriosclerosis, in which the same symptoms appeared, and in which the disturbance with the ophthalmoscopic changes disappeared very quickly.

The value of X-ray pictures of the accessory sinuses, especially of the antrum, is estimated differently by various authors. Many claim that at times they give rise to errors, and that the exploratory puncture is better to be relied upon.

VOGEL (62, **Diagnosis of diseases of the accessory sinuses**) reports a case which goes to show that while the roentgenograph is a valuable aid, it is not absolutely certain in the matter of diagnosis.

V.—THE LIDS AND LACRIMAL ORGANS.

64. BIRCH-HIRSCHFELD. **A simple operation for paralytic ectropion and spastic entropion.** *Klin. Monatsbl. f. Augenheilk.*, lxvii., p. 265.

65. CLAPP, C. A. **Entropion following influenza, with new surgical procedure.** *American Journal of Ophthalmology*, July, 1922.

66. COMBERG. **Inflammatory ptosis.** *Zeitschr. f. Augenheilk.*, xlvi., p. 249.

67. ESSER. **Shifting of the canthus.** *Arch. f. klin. Chir.*, cxv., p. 704.

68. FEIGENBAUM. **Tarsectomy and its curative value in trachoma and trichiasis.** *Klin. Monatsbl. f. Augenheilkunde*, lxvii., p. 600.

69. FRANCKE. **Two cases of streptothrix in the canaliculi.** *Ibid.*, p. 444.

70. GERNET. **Excision of the tarsus.** *Zeitschr. f. Augenheilk.*, xlvi., p. 285.

71. KUHNT. **Treatment of lagophthalmos.** *Ibid.*, xlvi., p. 46.

72. LANE, LAURA A. **A study of tumors of the lacrimal gland with report of a mixed tumor.** *American Journal of Ophthalmology*, June, 1922.

73. LANGE. **Results of Toti's operation.** *Deutsche. med. Wochenschrift*, xlvii., p. 1557.

74. LOEHLEIN. **Spirochætæ and bacillus fusiformis in dacryocystitis.** *Archiv. f. Augenheilkunde*, lxxxix., p. 201.

75. OHM. **Report of seventy Toti operations.** *Zeitschr. f. Augenheilk.*, xlvi., p. 37.

76. PIESBERGEN. **Polyp in the lacrimal sac.** *Klin. Monatsbl. f. Augenheilk.*, lxvi., p. 695.

77. RICHTER. **West's operation.** *Ibid.*, lxvii., p. 114.

78. RUTTIN AND NOWAK. **West's operation.** *Ibid.*, lxvii., p. 323.

79. WEINBERG. **Gangrene of the lid with diphtheria bacilli.** *Archiv. f. Ophthalmologie*, civ., p. 345.

BIRCH-HIRSCHFELD (64, **A simple operation for paralytic ectropion and spastic entropion**) says that the following operation gives good results. The orbicularis is laid bare by an incision 3*mm* from the margin of the lid, and the upper part of this muscle is dissected up from the underlying tissue for a breadth of 3*mm*. Two threads are passed close together through the muscle bundles, the muscle is cut between the threads, and then, after crossing, is sutured to the skin, in such a way that the temporal muscle bundles are attached to the nasal, and the nasal muscle bundles to the temporal portion of the lid skin. The wound is then closed with sutures.

CLAPP'S (65, **Entropion following influenza, with new surgical procedure**) patient was a child twenty-seven months old which, when four months of age developed entropion so that

the lashes brushed over the cornea. The condition was caused by a gradual absorption of the orbital fat. He did a fat transplantation, with partial success, by dissecting into the orbit through the skin of the lid. Excision of the skin later completed the cure. The child, before the eye trouble began, had a severe attack of influenza which he holds responsible for the trouble.

FEIGENBAUM (68, **Tarsectomy and its curative value in trachoma and trichiasis**) recommends this as an excellent operation, especially in regions where trachoma is endemic. Among its advantages may be enumerated protection and reparation of the endangered cornea, correction of entropion and trichiasis, and the freeing of the patients as quickly as possible from their trouble and allowing them to return to work. One of the effects he has often noticed is the extensive clearing up of pannus. Tarsectomy with suturing of the tendon of the levator he thinks a good operation for ptosis.

GERNET (70, **Excision of the tarsus**) also considers tarsectomy an excellent treatment for trachoma, especially in trachomatous regions. One unpleasant consequence is occasionally the formation of a sort of gable in the middle of the upper lid. This frequently disappears of itself and does not call for intervention until there appears to be no hope for later improvement. It is probably caused by the involvement of the tendon of the levator in the scar. Tarsectomy is also a good operation for entropion.

COMBERG (66, **Inflammatory ptosis**) is of the opinion that the swelling of the tissues and the diminution of mobility are the most important factors in the production of inflammatory ptosis. Another important factor may be the increase in the muscular tonus of the inflamed tissue, as the predominance of the tonically contracted orbicularis favors ptosis. In chronic inflammations the increase of volume may produce a ptosis, and in these cases the diminution of elasticity in the tissues assists.

ESSER (67, **Shifting of the canthus**) describes an operation for elevating a canthus which has been drawn down by a cicatrix in the lower lid. A pedicled flap is taken from the upper lid, its size proportioned to the effect desired, with its pedicle close to the canthus. An incision is then made parallel

to the intermarginal portion of the drawn down lid, sufficiently deep to give good mobilization to the canthus and the intermarginal portion. This incision may be very difficult to make because of the cicatrization. The pedicled flap is placed in the wound surface thus formed. Long threads are introduced into the margin of the wound between the intermarginal portion and the implanted flap and fastened above in the brow, so that an upward tension is exerted.

KUHNT (71, **Treatment of lagophthalmos**) has thought out a new procedure, whereby the lower lid may be fixed and the upper lid held tensely down. This is done by means of threads which start from the internal ligament as a base, are anchored in the superficial layers of the tarsus, and are fastened to the periosteum of the temporal margin of the orbit. An incision 3 to 4*mm* long is made over the internal ligament, then four parallel incisions of the same length some 2 to 2.5*mm* from the margin of the upper lid down to the tarsus, the inner and outer being over the ends of this tissue, four similar incisions in the lower lid only 2*mm* from its margin, and finally an incision 3*mm* long down to the periosteum 3*mm* beyond the outer canthus. A moderately strong thread armed with two needles is then introduced in the following way: One needle is passed upward, the other downward through the middle of the internal ligament, the thread drawn tight and the two parts crossed so that the upper portion of the thread goes to the lower lid, the lower to the upper. The needles are then passed subcutaneously to the first incision in each lid, from which the thread is at first allowed to hang in loops. Then the needles are passed subcutaneously to the second incision, taking in the superficial layers of the tarsus on the way, again allowing the thread to hang in loops. This is repeated to the next incision, and finally both parts of the thread are brought out in the incision outside of the canthus. Here they are crossed again and attached to the periosteum. The threads are now drawn tight, beginning at the nasal end between the incisions; the loops disappear and the lids are fixed by the subcutaneous thread. The palpebral fissure should then be a couple of millimeters narrower than that of the sound eye. The operation is completed by closing the incisions with sutures, fixation of the lids by vertical strips of adhesive plaster, and a dressing.

WEINBERG (79, **Gangrene of the lid with diphtheria bacilli**) reports two cases of infants with an ulcer in the canthus which did not respond to local treatment. In one case there was nasal diphtheria, in the other croupous conjunctivitis. Common to both were the presence of diphtheria bacilli, the involvement of the cornea, the good general condition, the slight elevation of temperature, and the good effect of serum. Three other cases were collated from literature. In all five the disease began soon after birth with a gangrene of the skin, so a vaginal infection is probable. The cornea was regularly involved, while there was little disturbance of temperature and the general condition remained good.

FRANCKE (69, **Two cases of streptothrix in the canaliculi**) reports two cases, one of which is notable inasmuch as the upper canaliculus was the one involved, which is rather unusual, and as a cure was secured by repeatedly pressing out the contents of the canaliculus without slitting it.

LANE (72, **Tumors of the lacrimal gland**) has reviewed the literature of the subject very thoroughly and collected the reports of 229 cases excluding Mikulicz' syndrome which is not a true neoplasm. A little less than one half the growths were of mixed type and there is a strong tendency toward malignancy. She recites one case of her own which when examined under the microscope showed in different areas glandular epithelium resembling adeno-fibroma, hyaline changes and myxomatous appearance suggesting myxosarcoma. A full bibliography is appended.

LOEHLEIN (74, **Spirochætæ and bacillus fusiformis in dacryocystitis**) found these microörganisms in the thin, purulent, fœtid secretion of a patient 25 years old. The dacryocystitis was possibly to be ascribed to a traumatism received years before that had injured the bone. For a year the pus had no longer drained into the nose and had acquired a fœtid odor.

PIESBERGEN (76, **Polyp in the lacrimal sac**) found a pedicled growth springing from the nasal wall of the lacrimal sac, and fairly filling the distended sac. Histologically it was composed of young, very cellular connective tissue with numerous leucocytes and plasma cells. The wall of the sac presented a thickening of connective tissue at the place of origin of the

pedicle, elsewhere was almost normal. The growth was free from epithelium, though on its upper surface numerous connective tissue cells simulated a coat of epithelial cells in one or two layers. Six or eight weeks previously the eye had presented a purulent inflammation and the lacrimal sac had been washed out twice and probes passed. An acute dacryocystitis brought the patient under observation.

LANGE (73, **Toti's operation**) reports the results in 29 cases, not selected, but some given preparatory treatment. A good result was obtained in twenty-three.

OHM (75, **Toti's operation**) reviews seventy of these operations. The ages of the patients varied from 5 to 62 years. Most of them were suffering from purulent dacryocystitis. Previous slitting of the canaliculus is not a contraindication. General anæsthesia is to be preferred. The results were after three months, 81.6% cures, 18% failures. The cures were demonstrated by washing through the canal and by the fluorescin test. Three fistulæ of the lacrimal sac healed quickly after this operation had been performed.

RICHTER (77, **West's operation**) considers this operation a distinct advance because it furnishes a good outflow, leaves behind no epiphora, and does not call for removal of the lacrimal gland. Further, it does away with probing, and strictures are not to be feared. The operation is made easier by the introduction of a probe into the lacrimal canal. A broad removal of the bony wall and soft parts is an essential to success. There is only a little pain associated with the operation. After treatment is conducted through the nose. In the discussion of this paper Clausen said that he considered ectatic lacrimal sacs as particularly suited to West's operation, while a purulent condition of the sac seemed to him almost always a contraindication. He thinks Toti's operation easier and the field of operation clearer. After both operations a daily irrigation is necessary at first, once or twice a week later.

RUTTIN and NOWAK (78, **West's operation**) report their observations separately. Ruttin reported nineteen cases with ten cures, the time of observation being from four to thirty-six months. The operation is difficult when the root of the nose is broad. The prognosis is best when the lacrimal sac is bluish gray, worse when it is yellow. Nowak found that

the operation gives good results in blennorrhea with ectasia of the lacrimal sac and simple stenosis; he had observed three failures in dacryocystitis with thickening of the walls. The opening made by the operation diminishes in the course of time without compromising the outflow of tears. The conjunctiva becomes clean bacteriologically very soon. He had obtained cures in seventy per cent. of his cases.

VI.—THE MOTOR APPARATUS OF THE EYE.

80. AXENFELD. **Tenotomy of both internal recti at one sitting for convergent strabismus.** *Ophthalm. Society of Vienna*, August 4–6, 1921.

81. BIELSCHOWSKY. **Genesis of dissociated vertical movements.** *Ibid.*

82. BIELSCHOWSKY. **Operative treatment of vertical deviations of the eyes.** *Archiv f. Ophthalmologie*, cv., p. 656.

83. FREYTAG. **Congenital retraction movements of the eyeball.** *Augenaerztl. Vereinig. in Leipsic*, April 17, 1921.

84. GRIESSMANN. **Caloric stimulation of the labyrinth of the ear.** *Muenchener med. Wochenschrift*, li., p. 1648.

85. PURTSCHER. **A new advancement of the external rectus.** *Klin. Monatsbl. f. Augenheilkunde*, October, 1921, p. 454.

86. ROESSLER. **The blind spot in squinting eyes.** *Archiv f. Ophthalm.*, cv., p. 48.

87. RUMBAUR. **A case of bilateral voluntarily dissociated movements of the eyes.** *Vereinig d. Augenaerzte Schlesiens u. Posen*, April 30, 1921.

88. SAENGER. **Cortical localization of the lateral movements of the eyes.** *Deutsche Zeitschrift f. Nervenheilkunde*, lxx., p. 92.

89. SCHUSTER, P. **Pathology of vertical paralysis of the eye.** *Ibid.*, lxx., p. 97.

90. STOERK. **Is the aural reflex a pathognomonically valuable symptom?** *Zeitschrift f. Laryngologie*, x., p. 417.

91. ZIEGLER S. L. **Capsulomuscular advancement without incision.** *American Journal of Ophthalmology*, April, 1922.

AXENFELD (80, **Tenotomy of both internal recti for convergent strabismus**) emphasizes the fact that a preponderating adduction of both eyes is frequently present in convergent strabismus. In such cases a double tenotomy of the interni at one sitting is permitted if one works carefully and introduces a safety suture through the detached tendon. In such cases Axenfeld claims that he has obtained good stereoscopic vision. He makes the tenotomy subconjunctivally through a small incision which may be enlarged if necessary. A bandage is

applied for only one day. Like every method of treating strabismus this one demands individualization and the taking into consideration of the special conditions in each case. The advantages possessed by double tenotomy over advancement comprise less loss of time, less troublesome convalescence, and a quickly pleasing result. The possibility of an exceptional secondary squint is not proportioned to the gain.

PURTSCHER (85, **A new advancement of the external rectus**) describes an operation of which the essential feature seems to be that the muscle is drawn through a buttonhole in the insertion of its own tendon. Two double armed sutures are introduced, one 9*mm* behind the insertion, the other farther back through the belly of the muscle. The tendon is divided just in front of the first suture, thus some 7 or 8*mm* behind the insertion, the stump is seized with Prince's forceps and a buttonhole made through which the two ends of the anterior suture are passed and the posterior stump of the tendon drawn through. This suture is then anchored to the sclera one or two millimeters from the limbus. The posterior suture is likewise fastened to the sclera at the insertion of the tendon, securing the conjunctiva at the same time. The effect can be varied by the distance between these sutures.

ZIEGLER (91, **Capsulomuscular advancement without incision**) passes one end of a double armed suture through the conjunctiva and upper third of the muscle near its attachment and out again at the edge repeating the process 2*mm* farther back thus forming a whip-stitch. The other end of the suture is passed in similar manner through the lower third of the muscle. Each needle is then passed back under the conjunctiva and Tenon's capsule along the edge of the muscle for about 10*mm* and out through the conjunctiva. Each end of the suture is now firmly attached to the sclera near the corneal margin and when it is tightened the muscle, capsule, and conjunctiva are crumpled into a double loop.

Dissociated vertical movements may be either bilateral or monolateral. BIELSCHOWSKY (81, **Genesis of dissociated vertical movements**) shows that an alternating upward squint may be changed into an alternating downward one by placing a dark glass before the fixing eye, when the eye that turned up will turn down. This is evidence that the dissociated

strabismus is of nervous origin. While monolateral vertical movements can be made manifest usually only by covering, or greatly darkening, one eye, in some cases the effect can be obtained by illuminating one eye more strongly than the other. In this way atypical deviations of the dissociated vertical movements can arise.

BIELSCHOWSKY (82, **Operative treatment of vertical deviations**) says that the commonest cause of paretic vertical deviations is paralysis of the superior oblique. As advancement of the tendon of this muscle is impracticable, he performs tenotomy of the inferior rectus of the other eye, and guards against an over-effect by means of a suture. A double armed suture is passed through the tendon and then through a place in the conjunctiva, chosen according to the amount of effect desired; the tendon is then cut and the wound in the conjunctiva closed. The suture passed through the tendon is not tied until after the concurrent action of the two eyes has been tested. When the effect is not too great it is left untied and, an over-effect being excluded, may be removed the next day. The rare paralysis of the inferior oblique hardly calls for operative correction because the subjective troubles are slight. As regards the treatment of nonparetic vertical deviations, the conditions are simplest when simply a cosmetic effect is desired, which may be secured by properly performed tenotomies and advancements. Greater difficulties are presented when good vision is present in both eyes and there are troubles of fusion, for in these the vertical deviation may be manifest only at times.

SCHUSTER (89, **Pathology of vertical paralysis of the eye**) describes four cases. In all voluntary movement of the eye, in one downward, in three upward, were impossible. Yet the movement of the eyes upward on closure of the lids was preserved. In the case in which the paralysis was downward the patient could follow an object with his eyes, produced a nystagmus downwards, and when his head was thrown back his eyes went down. In a second case there was a slight movement upwards of the eyes when the head was bent forward. The points for investigation in all such cases are the voluntary glancing movement, fixation on a still object, fixation of a sudden stimulation of the retina with light, following of a

moved object, reaction of the eyes to acoustic stimulation, movements of the eyes during passive movements of the head, and the development of nystagmus. The trouble seems to be located by the writer in the region of the corpora quadrigemina.

SAENGER (88, **Cortical localization of the lateral movements of the eyes**) discusses the findings at autopsy of quite a number of cases. In twelve cases the trouble was in the frontal portion of the brain, in four in the frontal and temporal lobes, in fourteen the parietal lobes also were involved, in six the parietal lobes alone, in five the temporal lobes alone, in ten the occipital lobes were affected, but never alone, always with involvement of some other portion of the brain. In eighteen the trouble was in the large ganglia or in the internal capsule. When one considers that opposed to these variable conditions are numerous negative cases one is led to the conclusion that conjugate deviations are of no special value for diagnosis.

RUMBAUR (87, **Bilateral voluntarily dissociated movements of the eyes**) reports the case of a 16 years old girl who could look straight ahead with one eye and voluntarily turn the other in to an extreme degree. A strong accommodation of both eyes was shown by the contraction of the pupils and skiascopically.

FREYTAG (83, **Congenital retraction movements of the eyeball**) observed these in a man 17 years old. There was a slight asymmetry of the face, left enophthalmos of *2mm* with narrowing of the palpebral fissure. Aside from medullated nerve fibers the left eyeball was normal. The adduction of the left eye was slight but replaced by a sinking or retraction of *5mm*. Passive abduction and adduction of the eye were good. The cause was thought to be an abnormal insertion of the internus too far back.

ROESSLER (86, **The blind spot in squinting eyes**) finds that in normal eyes the blind spot is of a certain position, that exceptions to this rule occur in only a small percentage and then only with the limits of five or six degrees, while in squinting eyes a considerable displacement is demonstrable as a rule, in 68 out of his 78 cases. He also finds that it is just the cases with normally placed blind spot that a therapeutic effect of operation can be expected, *i.e.*, a useful binocular vision can be obtained. Thus a certain prognosis possibly may be ob-

tained by means of the blind spot. The displacements found varied from twenty to thirty degrees above and below the normal position. The deviation was not confined to the squinting eye, but almost without exception was present in the other also. Excentric fixation did not come into question, as was shown by the eyes with $\frac{6}{18}$ vision or better. Binocular single vision could not be obtained in any of these cases. Roessler thinks the simplest and most probable explanation is a displacement of the nervous elements of the retina, a heterotopia of the entrance of the optic nerve, or of the fovea centralis. But in some cases a fault of the motor apparatus must be supposed, as a laterally tilted position of the eyeball which is to be looked upon as a trace of a rotator paralysis that has passed over into a concomitant strabismus. A final explanation of his results he does not give, but he inclines to the opinion that there is a pathological distribution of the retinal end elements which induces a discrepancy in the binocular impression of vision, which in the lower degrees weakens, in the higher degrees prevents fusion. This perversion of the eyes is double.

GRIESSMANN (84, **Caloric stimulation of the labyrinth of the ear**) maintains that it is not necessary to irrigate the ear in order to excite nystagmus. He claims that it is necessary only to introduce a bit of cotton wet with cold or hot water into the external auditory meatus in order to induce this phenomenon. So will hot or cold compresses applied to the concha of the ear, the throat or the neck.

STOERK (90, **Is the aural lid reflex a pathognomonically valuable symptom?**) thinks that this reflex has scarcely any diagnostic value whatever.

VII.—INTRAOCULAR TENSION AND GLAUCOMA.

92. BUETTNER. **Relations between trigeminal neuralgia and glaucoma.** *Archiv f. Augenheilkunde*, lxxxviii., p. 204.

93. CORDS. **Papillitis and glaucoma.** *Arch. f. Ophthalm.* cv., p. 916.

94. DUNN, J. **Observations concerning the causation of simple glaucoma.** *American Journal of Ophthalmology*, May, 1922.

95. FAITH, T. **Clinical observations on increased intraocular tension.** *Ibid.*, May, 1922.

96. GILBERT. **Examination of the aqueous.** *Arch. f. Augenheilk.*, 1921.

97. GROSZ. **Some methods of operation at the Hungarian university eye clinic.** *Archiv f. Ophthalmologie*, cv., p. 1075.

98. GROSZ. **Treatment of glaucoma.** *Zeitschr. f. Augenheilk.* xlv., p. 385.

99. GROSZ. **Expulsive hemorrhage after cyclodialysis.** *Ibid.*, p. 356.

100. HAMBURGER. **Tonometric studies of the nutrition of the eye in general and local diseases.** *Klin. Monatsbl. f. Augenheilk.* November-December, 1921.

101. IMRE. **Regulation of intraocular tension.** *Zeitschrift f. Augenheilkunde*, xlv., No. 5, 1921.

102. KAYSER. **Primary lacunar atrophy of the optic nerve or glaucomatous excavation.** *Klin. Monatsbl. f. Augenheilkunde*, lxvi., p. 923.

103. KOELLNER. **Glaucoma simplex with normal tonometer readings.** *Archiv f. Augenheilkunde*, lxxxix., p. 80.

104. POMPLUN. **Two cases of plexiform neuroma of the trigeminus with elephantiasis of the lids and hydrophthalmos in one.** *Klin. Monatsbl. f. Augenheilkunde*, lxvii., p. 242.

105. REIS. **Ophthalmomalacia.** *Arch. f. Ophthalm.* cv., p. 617.

106. RENYI. **Histology of glaucoma.** *Zeitschr. f. Augenheilk.*, xlv., p. 324.

107. STEINERT. **Experiences with Elliot's trephining in the Leipsic clinic.** *Klin. Monatsbl. f. Augenheilkunde*, lvi., p. 924.

108. WERNICKE. **Glaucoma as an expression of intraocular sclerosis disseminata.** *Buenos Aères*, 1921.

109. WESSELY AND HOROVITZ. **Behavior of the tension of the eye in fever.** *Archiv f. Augenheilkunde*, lxxxix., 1921.

110. WOLF. **Morphology of the aqueous.** *Ibid.*, xc., 1921.

BUETTNER (92, **Trigeminal neuralgia and glaucoma**) reports the case of a woman 45 years old who for several years had suffered occasional attacks of pain in all three branches of the trigeminus. The pain usually started in the fall, reached its maximum in December, and passed away in February. In October, 1918, examination revealed no signs of glaucoma, yet in December of the same year glaucoma was present in both eyes. Trephining was performed on her right eye, iridectomy on her left. Since then there have been no more attacks of neuralgic pain. Hence it is thought that the previous attacks of neuralgia were signs of glaucoma, in spite of the fact that the one examination made did not reveal any objective symptoms.

CORDS (93, **Papillitis and glaucoma**) says that an œdema of the papilla appears during or immediately after an attack of glaucoma, and that a secondary œdema of the papilla is not

rare in hemorrhagic glaucoma. A choked disk is incapable of causing increase of tension, and glaucoma is almost never observed immediately after a papillitis, but some time after a papillitis glaucoma appears rather frequently. Glaucoma does not accompany such other troubles of the optic nerve as tuberculosis, syphilis, and tumor. The swelling of the papilla after or coincidently with the attack of glaucoma may be ascribed in part to the evening up of the excavation after reduction of the intraocular tension and the saturation of the tissue with œdema, in part to the drawing in of the retina. He says that a retinal detachment may fill up a glaucomatous excavation. When there is total atrophy of the nerve fibers and a deep excavation inflammatory processes can hardly account for the filling of the latter. It is when a part of the nerve fibers are present that a choked disk can be associated with an excavation. Glaucoma has sometimes been observed after albuminuric papilloretinitis. Here we may have to deal with an accumulation of albuminuric changes about the optic nerve, or an increase of pressure within the skull, or a disease of the optic nerve itself. Vascular changes are never absent in these cases. Glaucoma occurs in tuberculous vascula diseases in young people in which the little branches, sometimes the trunk of the central vein, is involved, giving the clinical picture of papillitis or choked disk, thrombosis of the central vein, or optic atrophy from nutritive disturbance, but it is chiefly in the cases with thrombosis of the veins that glaucoma is observed. Papillitis or choked disk may follow hemorrhages, and subsequently glaucoma.

DUNN (94, **Observations concerning the causation of simple glaucoma**) maintains that glaucoma is produced by swelling of the vitreous. Such a condition being compared to swelling of the lens and depending upon the same cause, age changes and undetermined state of the blood.

FAITH (95, **Clinical observations on increased intraocular tension**) speaks of the difficulty in differentiating between glaucoma and uveitis with plus tension. He thinks that the age of the patient, the absence of prodromal symptoms, the involvement of one eye only, lack of corneal haziness, and deep anterior chamber should be taken into consideration. If tension is complicating a uveitis, atropine is indicated. He

believes that some of the cases which are treated with pilocarpine and iridectomy belong in this category and would be relieved by the early use of a mydriatic.

GILBERT (96, **Examination of the aqueous**) examined the aqueous of healthy and diseased eyes first for their cell content, second by the globulin test, and third by Wassermann's test. The number of cells in the aqueous of unirritated eyes is very small, not over 2 per *cmm* at most, and the newly formed aqueous in uninflamed eyes remains very poor in cells. In inflamed eyes on the contrary there are many cells, the number depending on the stage and intensity of the inflammation. Nonne's globulin test gives a negative result in normal aqueous, and also when tabetic or specifically neuritic optic atrophy is present. In inflamed eyes the test is positive, and a difference may be noted between cases of syphilitic and nonsyphilitic iritis as in the former the result of the ammonium sulphate test is regularly more pronounced than in the latter; yet the test may possibly only parallel the content of albumin. Wassermann's test is always negative in the healthy and inflamed eyes of nonsyphilitic persons, and also in syphilitics when the eye is not irritated. But it is positive in syphilitic inflammations of the anterior portion of the globe, more so as a rule in specific iritis that in interstitial keratitis. Also in nonspecific inflammations the Wassermann of the aqueous may be positive when the patient is syphilitic and has a strongly positive serum Wassermann. Therefore it is only in individual cases that a quantitative comparison between the strengths of the reaction in the aqueous and the blood may have a diagnostic value.

GROSZ (97, **Some methods of operation at the Hungarian university eye clinic**) reports for the past eight years 842 iridectomies, 48 sclerotomies, 41 cyclodialyses, 3 sclerectomies according to Lagrange, and 521 trephinings. In prodromal and acute glaucoma iridectomy is performed. Lagrange only in simple glaucoma, sclerotomy in glaucoma of children. Many trephinings for chronic inflammatory and acute glaucoma, in place of which cyclodialysis is now often performed. Three late infections after trephining. Trephining gives good service in just the cases in which iridectomy is apt to fail.

GROSZ (98, **Treatment of glaucoma**) advocates trephining for chronic inflammatory and simple glaucoma, yet he does not think the operation suited for all cases which are not fit for iridectomy, because the technique is not easy, the sharpness of the trephine is hard to control, incarceration of the iris is possible, and the ciliary body may be injured. Hitherto cyclodialysis has been used only in cases of degenerated glaucoma, but recently he has used it in other cases with good results. Iridectomy still remains indicated in prodromal and acute glaucoma with an elastic, not atrophic iris.

GROSZ (99, **Expulsive hemorrhage after cyclodialysis**) reports a case in which cyclodialysis was performed after a trephining had failed. A few days later a hemorrhage appeared in the anterior chamber. Attempts to remove this failed as a clot had formed, but soon it became absorbed. A month later hemorrhage was renewed. Posterior sclerotomy gave no relief but a protrusion appeared in the wound with prolapse of the choroid. The eye was rendered totally blind and had to be removed.

HAMBURGER (100, **Tonometric studies of the nutrition of the eye in general and local diseases**) finds that the tension of healthy eyes sink during fever. Also that the tension of an eye with inflammatory hyperæmia does not, as a rule, rise, but falls.

IMRE'S (101, **Regulation of intraocular tension**) studies concerning the regulatory action of the endocrine glands upon the intraocular tension started with the observation of a pregnant woman who, without known cause and in spite of a good general condition, developed a serious bilateral hypotony with secondary keratomalacia. He ascribed this hypotony to a hyperfunction of the hypophysis, and found low intraocular tension in two other cases of hyperpituitarism. He found that increased and decreased function not only of the hypophysis, but also of the thyroid, and of the ovaries exert influence over the intraocular tension. He obtained good results in a series of cases with glaucoma from treatment with thyroidin. When this did not act satisfactorily, or was productive of unpleasant symptoms, reduction of the tension was obtained by the administration of hypophysis tablets, or the injection of extracts of the testicles or ovaries.

KAYSER (102, **Primary lacunar atrophy of the optic nerve or glaucomatous excavation**) observed for over fifteen years a patient who had an abrupt excavation in one eye for twelve years with displacement of the vessels and total loss of color. The vision was good at first but had recently sunk to $\frac{6}{12}$ with normal tension and normal field. Miotics were of no use. In the family were several large physiological excavations of the optic nerve. One brother suffered from the same disease, which had been made considerably worse by iridectomy. Such cases, Kayser thinks, should not be considered glaucoma, but as suffering from a disease of the optic nerve itself. The excavation of the papilla cannot be accounted for by increased tension, and he believes it due to a primary lacunar atrophy.

KOELLNER (103, **Glaucoma simplex with normal tonometer readings**) reports a case of simple glaucoma in which a high tension at first became permanently low. The case was one of a man 71 years old with a tension of his right eye between 40 and 60*mm*, of his left between 30 and 40. Two years later the tension of the right was between 24 and 30*mm*, of the left between 19 and 2*mm*, although no treatment had been employed. The patient was then under observation for a week. In the meantime the glaucomatous excavation had become complete in the right eye, while the left papilla was normal. Hence there are cases with marked glaucomatous excavation in spite of normal tension in which a certain increase of tension existed a long time before.

POMPLUN (104, **Plexiform neuroma of the trigeminus with elephantiasis of the lids and hydrophthalmos**) reports a case of plexiform neuroma of the temple and upper lid with elephantiasis, but without hydrophthalmos. His second case was that of a girl 6 years old with a tumor like thickening over the right eyebrow which extended upward to the margin of the hair and sent processes as large as quills into the thickened lid. She had hydrophthalmos also. The growth with its processes was excised. Histologically it appeared to be a neurofibroma, a connective tissue hyperplasia of the peri- and endoneurium with an increase of the connective tissue beneath the skin. The eyeball was not removed, so no connection between the tumor and the hydrophthalmos was proved, but Pomplun

believes it would show nerves pathologically changed in the same manner.

During the war REIS (105, **Ophthalmomalacia**) observed a peculiar clinical picture which looked at first like a keratitis and then developed delicate subepithelial opaque stripes in the cornea with severe symptom of irritation. The stripes were generally vertical, often with others crossing at acute angles. The tension of the eyes was subnormal. Reis thinks the corneal changes were secondary, and of the nature of ophthalmomalacia. He maintains that it was not a true keratitis, but a formation of folds in Bowman's membrane with changes in the epithelia and neighboring corneal tissue. The diminution of the tension of the eye is the essential for the formation of folds in the cornea. Anatomical examination showed no changes which were explanatory of the hypotony, which must have originated from conditions outside the eyes. Bud-like formations along the stripes, composed of circumscribed accumulations of cells, make the picture resemble that of herpes, while the injuries to the epithelium depend on mechanical changes. The twenty-six cases reported were all of soldiers, only two of whom had not been in the field. In nearly all the eye had been injured. The subnormal tension seemed to rest on a neurotic basis, a view supported by the presence of various other neurotic disturbances of the general organism. Reis believes that the mental disturbances induced by the war was a precondition for the development of the disease, and that endogenous factors of a psychic-nervous variety exerted a peculiar influence upon the secretory nerves of the eye, which led to the decrease of tension with its consequences.

RENYI (106, **Histology of glaucoma**) says that there is a great proliferation of pigment in glaucoma, that the freed cells become deposited on the anterior surface of the iris, penetrate into it, break down here and are then to be found in the tissue spaces, the walls of the vessels, and in the epithelium. In absolute glaucoma many pigment cells and granules are to be found as far as the ciliary muscle, and also along and in the anterior ciliary vessels.

STEINERT (107, **Elliot's trephining**) gives the following statistics from the clinic in Leipsic. Trephined 429 eyes, of

which 179 were observed more than a year. Acute glaucoma, 83 per cent. with reduction of tension, 71.4 per cent. with improvement of vision. Chronic glaucoma; diminution of tension in 73.3 per cent., improvement of vision in 60.6 per cent. Visual field remained the same or was improved in 30 per cent. Simple glaucoma; reduction of tension in 69.2 per cent., improvement of vision in 57 per cent., of the field in 43.7 per cent. Cataract followed the operation in 17 cases. There were 12 late infections which caused the loss of six eyes. Trephining was not done in absolute glaucoma, but was in hydrophthalmos.

WERNICKE (108, **Glaucoma as an expression of intraocular sclerosis disseminata**) asks why, if sclerosis can cause choked disk, neuritis, and optic atrophy, it may not also manifest itself through an excavation. He considers Schnabel's caverns as probably foci of sclerosis. His explanation of the increase of tension is that microscopic foci of this disease attack the retinal portion of the ciliary body and the iris and cause an increase of tension through dispersion of pigment. He thinks the results of intervention support his theory. In sclerosis of the papilla, glaucoma simplex, no intervention is successful. When the foci are in the anterior part of the eye intervention may reduce the tension. The foci may then get well, or, in spite of normal tension, they may extend, when the vision will fail, or there may exist foci in the anterior segment and in the nerve at the same time. In the last case intervention may reduce the tension but will not affect the excavation. Objections to this theory are that it has not been proved, and that sclerosis is a disease of youth, while glaucoma is one of age. His answer to the last is that the severe cases of sclerosis die in youth, while the milder ones, which are not recognized as sclerosis, remain and later become glaucoma.

WESSELY and HOROVITZ (109, **Tension of the eye in fever**) have studied this subject partly in patients with malaria, partly in those who have received intragluteal injections of milk, and conclude that when in such rapidly running high fevers the blood pressure sinks, the tension of the eye follows the curve of the blood pressure exactly, with few exceptions.

WOLF (110, **Morphology of the aqueous**) finds the aqueous of healthy eyes extremely poor in cells. At the beginning of an attack of interstitial keratitis the number of cells is not very

high, under 100 per *cmm.* As the disease progresses the number of cells increase, lymphocytes and particularly leucocytes, and, at the height of the disease, iris cells. Specific treatment has no noticeable influence on the morphological picture. Iritis is marked by a great number of cells, 500 and more to the *cmm.* Endothelial cells from the cornea are those most frequently met with, contrary to the fact in parenchymatous keratitis, but they are better preserved, show fewer vacuoles and stain better. Next to the endothelia come lymphocytes, to which are added at the height of the disease leucocytes. Eosinophiles are never absent in syphilitic iritis, while in the non-specific forms they are usually present in fewer numbers. After the disease has lasted some time stroma cells of the iris, as well as ciliary and pigment epithelia are to be found. Occasionally the morphological findings in the aqueous may be useful in differential diagnosis.

(*To be continued.*)